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EQUINE NUTRITION & FEEDING

THIRD EDITION

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EQUINE NUTRITION AND FEEDING

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THIRD EDITION

DAVID FRAPE

PhD, CBiol, FIBiol, FRCPath



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Publishing

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Introduction to the Third Edition

The increased attention given to equine nutritional issues during the last 6–7 years by research groups around the world, has prompted me to revise the 2nd edition of this book. The preparation of this edition entailed the careful reading of the previous edition and with it the embarrassing discovery of a few errors, including one or two in equations, which I have now corrected.

It has been necessary to revise all chapters and other sections, some to a greater extent than others. The increased understanding of gastrointestinal tract function has led to a considerable number of changes to Chapters 1 and 2. The volume of work that has been undertaken with regard to skeletal growth and development (Chapters 7 and 8) has partly explained the mechanisms involved in endochondral ossification, but the story is incomplete. Work has been undertaken into the causes of several metabolic diseases (Chapter 11), but as yet their aetiology is obscure. The role of calcium in bone formation has been understood for many years, yet recent evidence has required that dietary needs be revised (Chapter 3). A similar situation has arisen with several vitamins and other minerals/trace minerals to which reference is made in Chapters 3 and 4. A brief account of several novel feeds, supplements and toxins is given and this has led to the extension of Chapter 5. Exercise physiology has continued to interest many research groups so that Chapters 6 and 9 have been revised. This has included a summary of procedures adopted, both historically and today, to measure energy consumption. Novel acronyms and terms have invaded scientific speech for which textual definitions are given.

A note on nomenclature: EC numbers have been used throughout when referring to specific enzymes. More detailed information about this system may be found in Chapter 12, p. 488.

Finally, I trust that an immanent characteristic of this 3rd edition is as a source reference for each of the more recent and important pieces of evidence in each of the areas covered. This may assist research workers and provide students with what I hope is a useful brief account upon which they might base their future activities;

but I must pay tribute to the authors of the papers upon which these pages have depended. Whereas valid disagreements in the literature have been aired, an eclectic set of references has, I hope, been distilled into a readable and comprehensible discourse.

David Frappe

Acknowledgements

I should like to thank Professor Franz Pirchner for reading and providing helpful comments on the amendments to Chapter 6 and to thank my wife, Margery, for her encouragement and support.

List of Abbreviations

AAT	aspartate aminotransferase
acetyl-CoA	acetyl coenzyme A
ACTH	adrenocorticotrophic hormone
ADAS	Agricultural Development and Advisory Service
ADF	acid detergent fibre
ADP	adenosine diphosphate
a.i.	active ingredient
AI	artificial insemination
ALP	alkaline phosphatase
ALT	alanine aminotransferase
AMP	adenosine monophosphate
AN	adenine nucleotides
AST	aspartate aminotransferase
ATP	adenosine triphosphate
BAL	bronchoalveolar lavage
BE	base excess
BFGF	basic fibroblast growth factor
BHA	butylated hydroxyanisole
BHT	butylated hydroxytoluene
BMR	basal metabolic rate
BSP	Bromsulphalein TM (sulphobromophthalein)
BW	body weight
CCO	cytochrome c oxidase
CF	crude fibre
CFU	colony-forming unit
CK	creatine kinase
COPD	chronic obstructive pulmonary disease
CP	crude protein
DCAB	dietary cation–anion balance
DCAD	dietary cation–anion difference

DCP	digestible crude protein
DDS	distiller's dark grains
DE	digestible energy
DM	dry matter
DMG	N,N-dimethylglycine
DMSO ₂	dimethylsulphone
DOD	developmental orthopaedic disease
ECF	extracellular fluid
EDM	equine degenerative myeloencephalopathy
EE	ether extract
EMND	equine motor neuron disease
ERS	exertional rhabdomyolysis syndrome
EU	European Union
EVH-1/4	equine herpesvirus
FAD	flavin adenine dinucleotide
FE	fractional electrolyte excretion
FFA	free fatty acid
FSH	follicle-stimulating hormone
FTH	fast twitch, high oxidative
FT	fast twitch, low oxidative
FTU	fungal titre unit
GE	gross energy
GGT	gamma-glutamyltransferase
GI	gastrointestinal
GLC	gas-liquid chromatograph
GnRH	gonadotropin-releasing hormone
GSH-Px	glutathione peroxidase
GSH	glutathione
Hb	haemoglobin
hCG	human chorionic gonadotropin
HI	heat increment
HPLC	high performance liquid chromatography
HPP	hyperkalaemic periodic paralysis
ICF	intracellular fluid
IGER	Institute of Grassland and Environmental Research
IMP	inosine monophosphate
INRA	Institut National de la Recherche Agronomique
iu	international unit
i.v.	intravenous
LBS	<i>Lactobacillus</i> selection
LDH	lactic dehydrogenase
LH	luteinizing hormone
LPL	lipoprotein lipase
LPS	lipopolysaccharides

MADC	matières azotées digestibles corrigées (<i>or</i> cheval)
MDA	malonyldialdehyde
ME	metabolizable energy
MSG	monosodium glutamate
MSM	methyl sulphonyl methane
NAD	nicotinamide adenine dinucleotide
NADP	nicotinamide adenine dinucleotide phosphate
NDF	neutral detergent fibre
NE	net energy
NEFA	nonesterified fatty acid(s)
NFE	nitrogen free extractive
NIS	nutritionally improved straw
NPN	non-protein nitrogen
NRC	National Research Council
NSC	non-structural carbohydrate
NSHP	nutritional secondary hyperparathyroidism
OC	osteocondrosis
OCD	osteocondritis dissecans
OM	organic matter
PAF	platelet activating factor
PCV	packed cell volume
PCr	phosphocreatine
PDH	pyruvate dehydrogenase
PN	parenteral nutrition
PTH	parathyroid hormone
PUFA	polyunsaturated fatty acid
RDR	relative dose response
RER	recurrent exertional rhabdomyolysis
RES	reticuloendothelial system
RH	relative humidity
RQ	respiratory quotient
RVO	recovered vegetable oil
SAP	serum alkaline phosphatase
SDH	sorbitol dehydrogenase
SET	standardized exercise test
SG	specific gravity
SGOT	serum glutamic–oxaloacetic transaminase
SID	strong ion difference
SOD	superoxide dismutase
ST	slow twitch, high oxidative
STP	standard temperature and pressure
T ₃	triiodothyronine
T ₄	thyroxine
TAG	triacylglycerol

TB	Thoroughbred
TBA	thiobarbituric acid
TBAR	thiobarbituric acid reactive substance
TCA	tricarboxylic acid
TLV	threshold limiting value
TPN	total parenteral nutrition
TPP	thiamin pyrophosphate
TRH	thyrotropin-releasing hormone
TSH	thyroid-stimulating hormone
UDP	uridine diphosphate
UFC	unité fourragère cheval
UKASTA	United Kingdom Agricultural Supply Trade Association
VFA	volatile fatty acid
VLDL	very low density lipoprotein
WBC	white blood cell; leukocyte

Chapter 1

The Digestive System

A horse which is kept to dry meat will often slaver at the mouth. If he champs his hay and corn, and puts it out again, it arises from some fault in the grinders . . . there will sometimes be great holes cut with his grinders in the weaks of his mouth. First file his grinders quite smooth with a file made for the purpose.

Francis Clater, 1786

Horses are ungulates and, according to J.Z. Young (1950), are members of the order Perissodactyla. Other extant members include asses, zebras, rhinoceroses and tapirs. Distinctive characteristics of the order are the development of the teeth, the lower limb with the peculiar plan of the carpus and tarsus bones and the evolution of the hind gut into chambers for fermentation of ingesta. Each of these distinctive features will play significant roles in the discussions in this text.

The domesticated horse consumes a variety of feeds ranging in physical form from forage with a high content of moisture to cereals with large amounts of starch, and from hay in the form of physically long fibrous stems to salt licks and water. In contrast, the wild horse has evolved and adapted to a grazing and browsing existence, in which it selects succulent forages containing relatively large amounts of water, soluble proteins, lipids, sugars and structural carbohydrates, but little starch. Short periods of feeding occur throughout most of the day and night, although generally these are of greater intensity in daylight. In domesticating the horse, man has generally restricted its feeding time and introduced unfamiliar materials, particularly starchy cereals, protein concentrates and dried forages. The art of feeding gained by long experience is to ensure that these materials meet the varied requirements of horses without causing digestive and metabolic upsets. Thus, an understanding of the form and function of the alimentary canal is fundamental to a discussion of feeding and nutrition of the horse.

THE MOUTH

Eating rates of horses, cattle and sheep

The lips, tongue and teeth of the horse are ideally suited for the prehension, ingestion and alteration of the physical form of feed to that suitable for propulsion through the gastrointestinal (GI) tract in a state that facilitates admixture with digestive juices. The upper lip is strong, mobile and sensitive and is used

during grazing to place forage between the teeth; in the cow the tongue is used for this purpose. By contrast, the horse's tongue moves ingested material to the cheek teeth for grinding. The lips are also used as a funnel through which water is sucked.

As distinct from cattle, the horse has both upper and lower incisors enabling it to graze closely by shearing off forage. More intensive mastication by the horse means that the ingestion rate of long hay, per kilogram of metabolic body weight (BW), is three to four times faster in cattle and sheep than it is in ponies and horses, although the number of chews per minute, according to published observations, is similar (73–92 for horses and 73–115 for sheep) for long hays. The dry matter (DM) intake per kilogram of metabolic BW for each chew is then 2.5 mg in horses (I calculate it to be even less) and 5.6–6.9 mg in sheep. Consequently, the horse needs longer daily periods of grazing than do sheep. The lateral and vertical movements of the horse's jaw, accompanied by profuse salivation, enable the cheek teeth to comminute long hay to a greater extent and the small particles coated with mucus are suitable for swallowing. Sound teeth generally reduce hay and grass particles to less than 1.6 mm in length. Two-thirds of hay particles in the horse's stomach are less than 1 mm across, according to work by Meyer and colleagues (Meyer *et al.* 1975b).

The number of chewing movements for roughage is considerably greater than that required for chewing concentrates. Horses make between 800 and 1200 chewing movements per 1 kg concentrates, whereas 1 kg long hay requires between 3000 and 3500 movements. In ponies, chewing is even more protracted – they require 5000–8000 chewing movements per 1 kg concentrates alone, and very many more for hay (Meyer *et al.* 1975b). Hay chewing, cf. pellets, by both horses and ponies, is protracted, with a lower chewing-cycle frequency, as the mandibular displacement is greater, both vertically and horizontally. Clayton *et al.* (2003) concluded, from this observation, that the development of sharp enamel points is more likely with a high concentrate diet.

Dentition

As indicated above, teeth are vital to the well-being of horses. Diseased teeth are an encumbrance. Primary disorders of the cheek teeth represented 87% of the dental disorders in 400 horses referred to Dixon *et al.* (2000a). The disorders included abnormalities of wear, traumatic damage and fractures from which the response to treatment was good.

Evidence has shown that abnormal or diseased teeth can cause digestive disturbances and colic. Apparent fibre digestibility, the proportion of faecal short fibre particles and plasma free fatty acids were all increased after dental correction of mares. Consequently, diseased teeth and badly worn teeth, as in the geriatric horse, can limit the horse's ability to handle roughage and may compromise general health. The apparent digestibility of the protein and fibre in hay and grain is reduced if the occlusal angle of premolar 307 is greater than 80° relative to the vertical angle (flattened) (Ralston *et al.* 2001). Infections of cheek teeth are not uncommon and

Dixon *et al.* (2000b) found that nasal discharge was more frequent with infections of caudal than with rostral maxillary teeth.

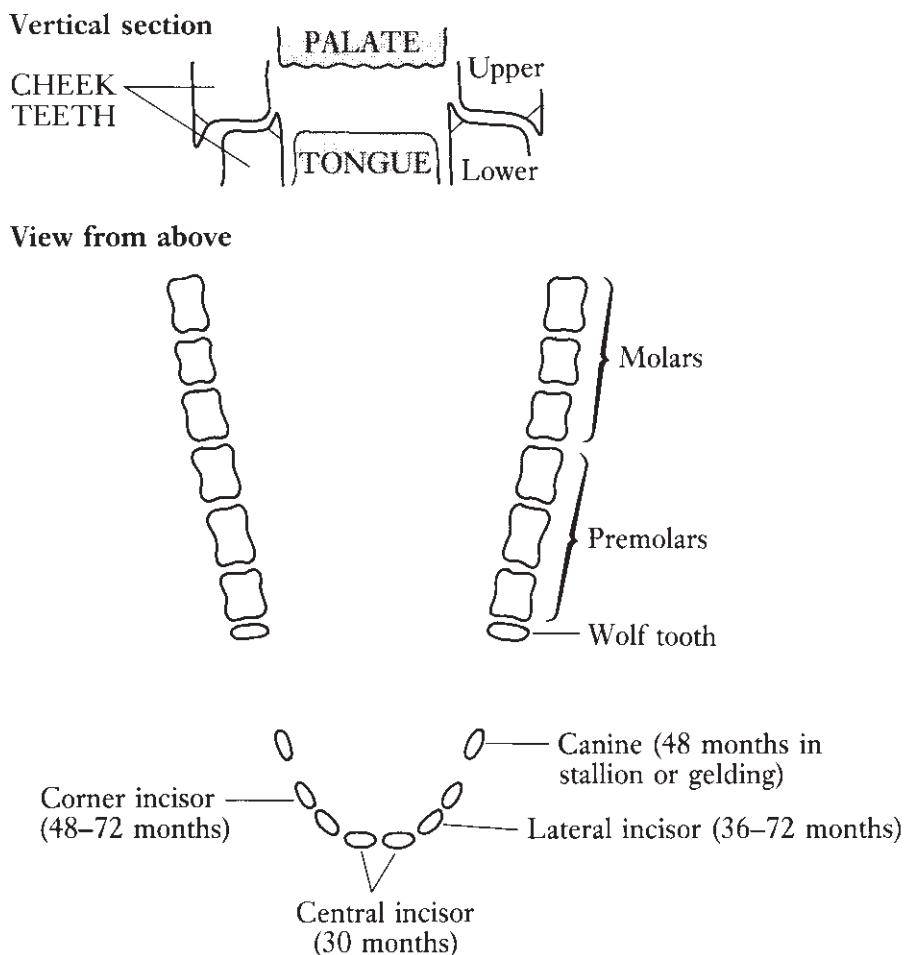
The normal horse has two sets of teeth. The first to appear, the deciduous, or temporary milk, teeth erupt during early life and are replaced during growth by the permanent teeth. The permanent incisors and permanent cheek teeth erupt continuously to compensate for wear and their changing form provides a basis for assessing the age of a horse. In the gap along the jaw between the incisors and the cheek teeth the male horse normally has a set of small canine teeth. The gap, by happy chance, securely locates the bit. The dental formulae and configuration of both deciduous and permanent teeth are given in Fig. 1.1. The lower cheek teeth are implanted in the mandible in two straight rows that diverge towards the back. The space between the rows of teeth in the lower jaw is less than that separating the upper teeth (Fig. 1.1). This accommodates a sideways, or circular, movement of the jaw that effectively shears feed. The action leads to a distinctive pattern of wear of the biting surface of the exposed crown. This pattern results from the differences in hardness which characterize the three materials (cement, enamel and dentine) of which teeth are composed. The enamel, being the hardest, stands out in the form of sharp prominent ridges. It is estimated that the enamel ridges of an upper cheek tooth in a young adult horse, if straightened out, would form a line more than 30cm (1ft) long. This irregular surface provides a very efficient grinding organ.

Horses and ponies rely more on their teeth than we do. People might be labelled concentrate eaters; concentrates require much less chewing than does roughage. Even among herbivores, horses and ponies depend to a far greater extent on their teeth than do the domesticated ruminants – cattle, sheep and goats. Ruminants, as discussed in ‘Eating rates of horses, cattle and sheep’, swallow grass and hay with minimal chewing and then depend on the activity of bacteria in the rumen to disrupt the fibre. This is then much more readily fragmented during chewing the cud.

Saliva

The physical presence of feed material in the mouth stimulates the secretion of a copious amount of saliva. Some 10–12l are secreted daily in a horse fed normally. This fluid seems to have no digestive enzyme activity, but its mucus content enables it to function as an efficient lubricant preventing ‘choke’. Its bicarbonate content, amounting to some 50mEq/l, provides it with a buffering capacity. The concentration of bicarbonate and sodium chloride in the saliva is, however, directly proportional to the rate of secretion and so increases during feeding. The continuous secretion of saliva during eating seems to buffer the digesta in the proximal region of the stomach, permitting some microbial fermentation with the production of lactate. This has important implications for the well-being of the horse (see Chapter 11).

Obstruction of the oesophagus by impacted feed or foreign bodies is not uncommon. To facilitate nutritional support during treatment of oesophageal perforation, a cervical oesophagotomy tube is placed and advanced into the stomach (Read *et al.*



Dental formula

$$\text{Deciduous : } 2(Di \frac{3}{3} Dc \frac{0}{0} Dp \frac{3}{3}) = 24$$

$$\text{Permanent : } 2(I \frac{3}{3} C \frac{1}{1} P \frac{3 \text{ or } 4}{3} M \frac{3}{3}) = 40 \text{ or } 42$$

Fig. 1.1 Configuration of permanent teeth in the upper or lower jaw (the molars and premolars in the lower jaw are slightly closer to the midline). The deciduous teeth on each side of each jaw are: three incisors, one canine, three molars. The deciduous canines are vestigial and do not erupt. The wolf teeth (present in the upper jaw of about 30% of fillies and about 65% of colts) are often extracted as their sharp tips can injure cheeks when a snaffle bit is used. Months (in parentheses) are approximate ages at which permanent incisors and canines erupt, replacing the deciduous teeth.

2002). An enteral diet includes an electrolyte mixture (partly to compensate for salivary electrolyte losses through the oesophagotomy site), sucrose (1.2 kg/d), casein, canola rapeseed oil (1.1 l/d) and dehydrated alfalfa pellets. A nasogastric tube is subsequently introduced to allow repair of the oesophagotomy site.

THE STOMACH AND SMALL INTESTINE

The first quantitative aspects of digestion were demonstrated by Waldinger in 1808 with the passage of capsulated feedstuff through the intestines. Intensive studies concerning the physiology of digestion were started in Paris around 1850 by Colin, but they proceeded predominately from 1880 in Dresden by Ellenberger and Hofmeister who investigated the mouth, stomach and small intestine. Scheunert continued with work on the large intestine in Dresden and Leipzig until the 1920s. Although the apparent digestibility of cellulose was appreciated by 1865 it took another 20 years for the discovery of the process of microbial digestion in the equine large intestine. Until 1950 most routine equine digestibility experiments were conducted in Germany, France and the USA (Klingeberg-Kraus 2001), while comparative studies were conducted by Phillipson, Elsdon and colleagues at Cambridge in the 1940s.

Development of the gastrointestinal (GI) tract and associated organs

The GI tract tissue of the neonatal foal weighs only 35 g/kg BW, whereas the liver is large, nearly in the same proportion to BW, acting as a nutrient store for the early critical days. By six months of age the GI tract tissue has proportionately increased to 60 g/kg BW, whereas the liver has proportionately decreased to about 12–14 g/kg BW. By 12 months both these organs have stabilized at 45–50 g/kg BW for the GI tract and 10 g/kg BW for the liver. Organ size is also influenced by the activity of the horse. After a meal, the liver of mammals generally increases rapidly in weight, probably as a result of glycogen storage and blood flow. In the horse the consumption of hay has less impact on liver glycogen, so that following a meal of hay the liver weighs only three-quarters of that following mixed feed. Moreover, during and immediately after exercise the GI tract tissue weighs significantly less than in horses at rest, owing to the shunting of blood away from the mesenteric blood vessels to the muscles. At rest about 30% of the cardiac output flows through the hepatic portal system. More about these aspects is discussed in Chapter 9.

Surprisingly, the small intestine does not materially increase in length from 4 weeks of age, whereas the large intestine increases with age, the colon doing so until 20 years at least. The distal regions of the large intestine continue extension to a greater age than do the proximal regions. This development reflects the increasing reliance of the older animal on roughage. In an adult horse of 500 kg BW the small intestine is approximately 16 m in length, the caecum has a maximum length of about 0.8 m, the ascending colon 3 m and the descending colon 2.8 m.

Transit of digesta through the GI tract

The residence time for ingesta in each section of the GI tract allows for its adequate admixture with GI secretions, for hydrolysis by digestive enzymes, for absorption of the resulting products, for fermentation of resistant material by bacteria and for the

absorption of the products of that fermentation. Transit time through the GI tract is normally considered in three phases, owing to their entirely different characteristics. These phases are:

- (1) expulsion rate from the stomach into the duodenum after a meal;
- (2) rate of passage through the small intestine to the ileocaecal orifice;
- (3) retention time in the large intestine.

The first of these will be considered below in relation to gastric disorders. Rate of passage of digesta through the small intestine varies with feed type. On pasture this rate is accelerated, although a previous feed of hay causes a decrease in the rate of the succeeding meal, with implications for exercise (see Chapter 9). Roughage is held in the large intestine for a considerable period that allows microbial fermentation time to break down structural carbohydrates. However, equine GI transit time of the residue of high fibre diets is less than that of low fibre diets of the same particle size, in common with the relationship found in other monogastric animals.

Digestive function of the stomach

The stomach of the adult horse is a small organ, its volume comprising about 10% of the GI tract (Fig. 1.2, Plate 1.1). In the suckling foal, however, the stomach capacity represents a larger proportion of the total alimentary tract. Most digesta are held in the stomach for a comparatively short time, but this organ is rarely completely empty and a significant portion of the digesta may remain in it for two to six hours. Some digesta pass into the duodenum shortly after eating starts, when fresh ingesta enter the stomach. Expulsion into the duodenum is apparently arrested as soon as feeding stops. When a horse drinks, a high proportion of the water passes along the curvature of the stomach wall so that mixing with digesta and dilution of the digestive juices it contains are avoided. This process is particularly noticeable when digesta largely fill the stomach.

The entrance to the stomach is guarded by a powerful muscular valve called the cardiac sphincter. Although a horse may feel nauseated, it rarely vomits, partly because of the way this valve functions. This too has important consequences. Despite extreme abdominal pressure the cardiac sphincter is reluctant to relax in order to permit the regurgitation of feed or gas. On the rare occasions when vomiting does occur, ingesta usually rush out through the nostrils, owing to the existence of a long soft palate. Such an event may indicate a ruptured stomach.

Gastric anatomy differentiates the equine stomach from that of other monogastrics. Apart from the considerable strengths of the cardiac and pyloric sphincters, almost half the mucosal surface is lined with squamous, instead of glandular, epithelium. The glandular mucosa is divided into fundic and pyloric regions (Fig. 1.2). The fundic mucosa contains both parietal cells that secrete hydrochloric acid (HCl) and zymogen cells which secrete pepsin, while the polypeptide hormone gastrin is secreted into the blood plasma by the pyloric region. The

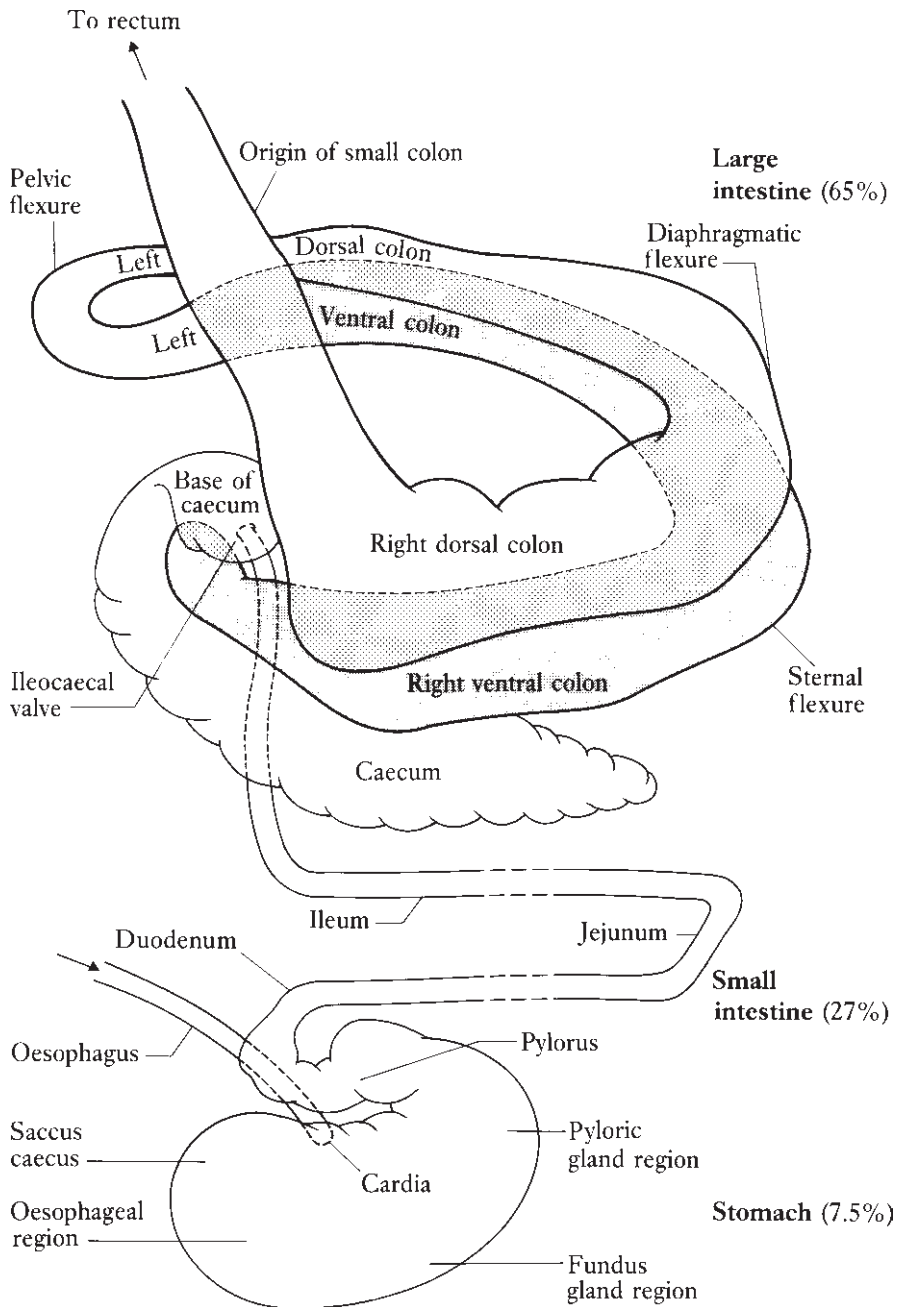


Fig. 1.2 GI tract of adult horse (relative volumes are given in parentheses).

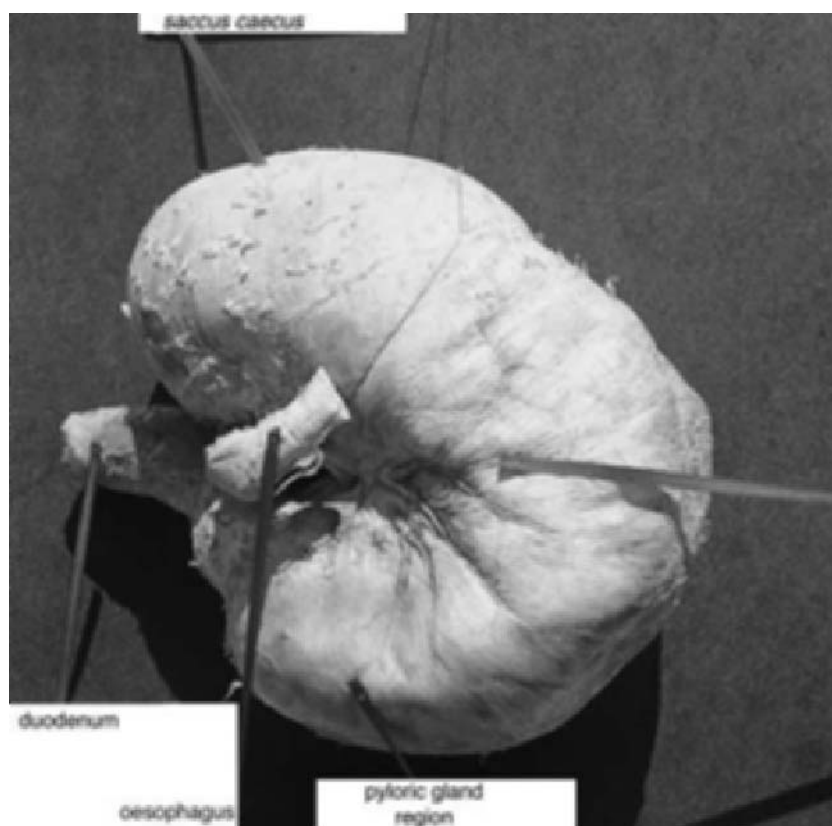


Plate 1.1 Stomach of a 550kg TB mare, capacity 8.4l, measuring about 20 × 30 × 15 cm. Acid fermentation of stomach contents takes place in the *saccus caecus* (top).

hormone's secretion is triggered by a meal, and equine studies in Sweden show that a mechanism of the gastric phase of release seems to be distension of the stomach wall by feed, but not the sight of feed. The greatest and most prolonged gastrin secretion occurs when horses eat hay freely (A. Sandin personal communication). In the horse gastrin does not seem to act as a stress hormone. The hormone strongly stimulates secretion of gastric acid and the daily secretion and release of gastric juice into the stomach amounts to some 10–30l. Secretion of gastric juice continues even during fasting, although the rate seems to vary from hour to hour.

HCl secretion continues, but declines gradually at a variable rate when the stomach is nearly empty and hence at that time the pH is around 1.5–2.0. The pH rises rapidly during a subsequent meal, especially that of grain only, partly as a consequence of a delay in gastrin secretion, compared with the more rapid gastrin response to hay. The act of eating stimulates the flow of saliva – a source of sodium, potassium, bicarbonate and chloride ions. Saliva's buffering power retards the rate at which the pH of the stomach contents decreases. This action, combined with a stratification of the ingesta, brings about marked differences in the pH of different regions (about 5.4 in the fundic region and 2.6 in the pyloric region).

Fermentation, primarily yielding lactic acid, occurs in the oesophageal and fundic regions of the stomach, but particularly in that part known as the *saccus caecus*, lined by the squamous cells. As digesta approach the pylorus at the distal end of the stomach, the gastric pH falls, owing to the secretion of the HCl, which potentiates the proteolytic activity of pepsin and arrests that of fermentation. The activity of pepsin in the pyloric region is some 15–20 times greater than in the fundic. Because of the stomach's small size and consequentially the relatively short dwell time, the degree of protein digestion is slight.

Gastric malfunction

Professor Meyer and his colleagues in Hanover (Meyer *et al.* 1975a) have made detailed investigations of the flow of ingesta and digesta through the GI tract of horses. In so far as the stomach is concerned their thesis is that abnormal gastric fermentation occurs when the postprandial dry matter content of the stomach is particularly high and a low pH is not achieved. There is, nevertheless, considerable layering and a differentiation in pH between the *saccus caecus* and pyloric region. Fermentation is therefore a normal characteristic of the region of higher pH and in that region the larger roughage particles tend to float. However, the dry-matter content, generally, is considerably lower following a meal of roughage than it is following one of cereals. After meals of 1 kg loose hay and 1 kg pelleted cereals the resulting gastric dry matters were, respectively, 211 and 291 g/kg contents.

The Hanover group compared long roughage with that which was chopped, ground or pelleted and observed that, as particle size of roughage was decreased, the gastric dry matter contents decreased from 186 to 132 g/kg contents and the rate of passage of ingesta through the stomach increased. The reason for this is probably that it is the finely divided material in a gastric slurry which passes first to the intestines. The slurry is forced into the duodenum by contractions termed *antral systole* at the rate of about three per minute. Nevertheless, it should be recalled that particle size is generally small as a result of comminution by the molars. With larger meals of pelleted cereal, up to 2.5 kg/meal, the gastric dry matter content attained 400 g/kg, and the pH was 5.6–5.8, for as long as two to three hours after consumption. The dry matter accumulated faster than it was ejected into the duodenum, and as cereals could be consumed more rapidly than hay, with a lower secretion of saliva, the dry matter of the stomach was higher following large meals of cereals. As much as 10–20% of a relatively small meal of concentrates (given at the rate of 0.4% BW) has been found to remain in the stomach six hours after feeding ponies. A high dry-matter content acts as a potent buffer of the HCl in gastric juice and the glutinous nature of cereal ingesta inhibits the penetration of cereal ingesta by those juices.

Together with the delay in gastrin release during a cereal meal, these factors could account for the failure of the postprandial pH to fall to levels that inhibit further microbial growth and fermentation. Lactic-acid producing bacteria (*Lactobacilli* and *Streptococci*) thrive (also see Probiotics, Chapter 5). Whereas *Streptococci* do

not produce gas, some *Lactobacillus* species produce carbon dioxide, thrive at a pH of 5.5–6.0 and even grow in the range 4.0–6.8, some strains growing in conditions as acid as pH 3.5. The pH of the gastric contents will even increase to levels that permit non-lactic-acid-producing, gas-producing bacteria to survive, producing large amounts of volatile fatty acids (VFAs). Gas production at a rate greater than that at which it can be absorbed into the bloodstream causes gastric tympany, and even gastric rupture, and hence it is desirable that the postprandial gastric pH falls sufficiently to arrest most bacterial growth and, in fact, to kill potential pathogens.

Gastric ulceration

The stratified squamous epithelial mucosa of the equine stomach exists in a potentially highly acidic environment and is susceptible to damage by HCl and pepsin. Bile, which is found in significant amounts in the stomach during long fasts, increases the risk of damage (Berschneider *et al.* 1999). Routine post-mortem examination of 195 Thoroughbreds (TBs) in Hong Kong (Hammond *et al.* 1986) revealed that 66% had suffered gastric ulceration. In TBs taken directly from training the frequency was 80%, whereas it was only 52% among those that had been retired for a month or more. The lesions seem to be progressive during training, but to regress during retirement. These lesions are not restricted to adult horses. Neonatal foals are able to produce highly acidic gastric secretions as early as two days old, and the mean pH of the glandular mucosal surface and fluid contents of 18 foals at 20 days old were 2.1 and 1.8, respectively (Murray & Mahaffey 1993). Ulceration and erosion occur in the gastric squamous mucosa, particularly that adjacent to the *margo plicatus*, as the squamous epithelial mucosa lacks the protective processes, especially the mucus–bicarbonate barrier, possessed by the glandular mucosa.

Observations by the research group in Hanover showed that clinical signs of periprandial colic and bruxism (grinding of teeth) were more pronounced in horses with the most severe gastric lesions of diffuse ulcerative gastritis. Their further evidence showed that ponies receiving hay only were free from lesions, whereas 14 out of 31 receiving concentrates had ulcerative lesions (see Chapter 11).

Although treatment with omeprazole, cimetidine or ranitidine, is effective, one must wonder whether infection plays a part in the equine syndrome (as it frequently does in man, where the organisms shrewdly protect themselves from acid by urease secretion with an acid pH optimum), as periprandial microbial activity and pH of gastric contents are higher in concentrate-fed animals. Moreover, the pH is lowest during a fast. If this proposal is true then quite different prophylaxis and treatment should be chosen.

Digestion in the small intestine

The 450 kg horse has a relatively short small intestine, 21–25 m in length, through which transit of digesta is quite rapid, some appearing in the caecum within 45 min

after a meal. Much of the digesta moves through the small intestine at the rate of nearly 30 cm/min. Motility of the small intestine is under both neural and hormonal control. Of a liquid marker instilled into the stomach of a pony, 50% reached the distal ileum in 1 hour, and by 1.5 hours after instillation 25% was present in the caecum (Merritt 1992 pers. comm.). The grazing horse has access to feed at all times and comparisons of quantities of feed consumed, where there is *ad libitum* access with similar quantities given following a 12-hour fast, showed that the transit of feed from stomach to the caecum is much more rapid following the fast.

To estimate transit time monofilament polyester bags with a pore size of 41 µm and containing 200 or 130 mg feed can be introduced into the stomach via a nasogastric tube and recovered in the faeces after transit times of 10 to 154 hours. Transit times and digestibility in the small intestine may be estimated following capture of the bags from near the ileocaecal valve with a magnet (Hyslop *et al.* 1998d). Caution should, however, be exercised in the interpretation of precaecal N-digestibility values, which can be considerably higher from the mobile bag cf. the ileal-fistula technique (Macheboeuf *et al.* 2003).

In consequence of the rapid transit of ingesta through the small intestine, it is surprising how much digestion and absorption apparently occur there. Although differences in the composition of digesta entering the large intestine can be detected with a change in diet, it is a considerably more uniform material than that entering the rumen of the cow. This fact has notable practical and physiological significance in the nutrition and well-being of the horse. The nature of the material leaving the small intestine is described as fibrous feed residues, undigested feed starch and protein, microorganisms, intestinal secretions and cell debris.

Digestive secretions

Large quantities of pancreatic juice are secreted as a result of the presence of food in the stomach in response to stimuli mediated by vagal nerve fibres, and by gastric HCl in the duodenum stimulating the release into the blood of the polypeptide hormone secretin. In fact, although secretion is continuous, the rate of pancreatic juice secretion increases by some four to five times when feed is first given. This secretion, which enters the duodenum, has a low order of enzymatic activity, but provides large quantities of fluid and sodium, potassium, chloride and bicarbonate ions. Some active trypsin is, however, present. There is conflicting evidence for the presence of lipase in pancreatic secretions, and bile, secreted by the liver, probably exerts a greater, but different, influence over fat digestion. The stimulation of pancreatic juice secretion does not increase its bicarbonate content, as occurs in other species. The bicarbonate content of digesta increases in the ileum, where it is secreted in exchange for chloride, so providing a buffer to large intestinal volatile fatty acids (VFA) (see 'Products of fermentation', this chapter).

The horse lacks a gall bladder, but stimulation of bile is also caused by the presence of gastric HCl in the duodenum. Secretion of pancreatic juice and bile ceases after a fast of 48 hours. Bile is both an excretion and a digestive secretion. As

a reservoir of alkali it helps preserve an optimal reaction in the intestine for the functioning of digestive enzymes secreted there. In the horse, the pH of the digesta leaving the stomach rapidly rises to slightly over 7.0.

Carbohydrates

The ability of the horse to digest soluble carbohydrates and the efficiency of the mucosal monosaccharide transport systems of the small intestine have been established by a series of oral disaccharide and monosaccharide tolerance tests (Roberts 1975b). This ability is important to an understanding of certain digestive upsets to which the horse is subject.

A high proportion of the energy sources consumed by the working horse contains cereal starches. These consist of relatively long, branched chains, the links of which are α -D-glucose molecules joined as shown in Fig. 1.3. Absorption into the bloodstream depends on the disruption of the bonds linking the glucose molecules. This is contingent entirely upon enzymes secreted in the small intestine. These are held on the brush border of the villi in the form of α -amylase (secreted by the pancreas) and as α -glucosidases (secreted by the intestinal mucosa) (see Table 1.1).

The secretions of the pancreatic juice release sufficient oligosaccharides for further hydrolysis by the brush border enzymes at the intestinal cell surface (Roberts

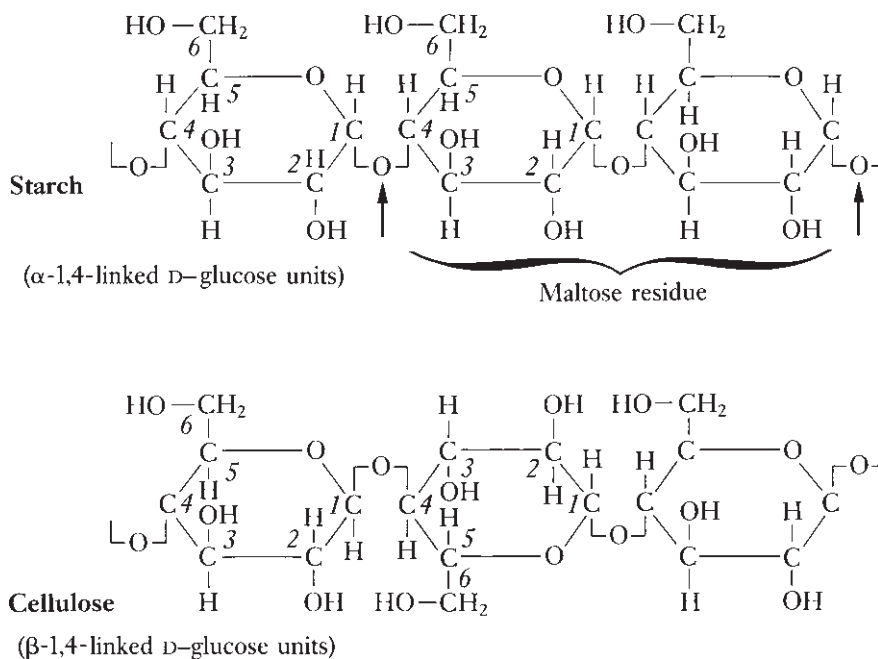


Fig. 1.3 Diagrammatic representation of three glucose units in two carbohydrate chains (the starch granule also contains amylopectin, which has both 1-4 linkages and 1-6 linkages). Arrows indicate site of intermediate digestion.

Table 1.1 Carbohydrate digestion in the small intestine.

Substrate	Enzyme	Product
Starch	α -Amylase	Limiting dextrins (about 34 glucose units)
Limit dextrins	α -Glucosidases (glucoamylase, maltase and isomaltase)	Glucose
Sucrose	Sucrase	Fructose and glucose
Lactose	Neutral- β -galactosidase (lactase)	Glucose and galactose

1975a). Active carrier mediated mechanisms then transport the final hexose products across the intestinal cell for uptake in the hepatic portal system. The digestive system can, however, be overloaded. Ponies weighing 266 kg BW were given 4 kg feed/day, as oat hulls:naked oats 2:1 (i.e. 1.33 kg naked oats). This led to changes in intracaecal fermentation, indicating that there was oat starch reaching that organ, although the intracaecal pH did not decrease below 6.5 (Moore-Colyer *et al.* 1997). Starch fermentation in the hind-gut and its consequences are discussed below and in Chapters 2 and 11.

The concentration of α -amylase in the pancreatic juice of the horse is only 5–6% of that in the pig, whereas the concentration of α -glucosidase is comparable with that in many other domestic mammals. The α -glucosidases (disaccharidases) include sucrase, the disaccharidase present in concentrations five times that of glucoamylase and capable of digesting sucrose. Sucrase activity is highest in the proximal small intestine and, whereas its activity is similar to that reported for other non-ruminant species, maltase activity is extremely high in comparison with that reported for other species. Maltase activity is expressed similarly in proximal, mid and distal regions. D-glucose and D-galactose are transported across the equine intestinal brush border membrane by a high affinity, low capacity, Na⁺/glucose cotransporter *type1* isoform (SGLT1) with rates of transport in the order, duodenum > jejunum > ileum (Dyer *et al.* 2002).

Another important disaccharidase in the intestinal juice is the β -glucosidase, neutral β -galactosidase (neutral or brush-border lactase), which is necessary for the digestion of milk sugar in the foal. This enzyme has a pH optimum of around 6.0. Whereas functional lactase is expressed all along the small intestine of the adult horse, the activity is less than that in the immature horse (Dyer *et al.* 2002), thus large quantities of dietary lactose may cause digestive upsets and adult horses are relatively lactose intolerant.

Healthy horses of all ages can absorb a glucose:galactose mixture without any change in the faeces. The relative intolerance is due to reduced lactose hydrolysis and does not normally involve the monosaccharide transport systems or malabsorption. If a suckling foal, or one given cow's milk, lacks an active form of the enzyme, it suffers from diarrhoea. An oral lactose tolerance test (1 g/kg BW as a 20% solution)

could be of clinical value to determine small intestinal mucosal damage in diarrhoeic foals, when the continued ingestion of lactose might be detrimental. The deficient digestion or malabsorption of carbohydrate, whether primary or secondary, can almost always be localized to a defect in the enzymic, or transport, capacity of the small intestinal surface cell (see Chapter 11).

Lindemann *et al.* (1983) gave adult horses lactose or maize starch at 2g/kg BW daily before a feed of wheat straw, or mixed with a diet of concentrate. Apparent precaecal digestibility of lactose was 38% and 71% in the straw and concentrate periods respectively and that of starch in those periods 88% and 93%. For straw about 1.2g and for concentrate 0.6g lactose per kg BW flowed into the caecum daily, leading to higher caecal VFA concentrations and a lower caecal pH with lactose than with starch in the straw period. Ileocaecal water flow reached 16.5 and 8.2kg/kg feed DM with lactose in the straw and concentrate periods respectively, compared with 15.2 and 7.0kg/kg with starch. The 38% and 71% apparent precaecal digestibility of lactose may partly reflect microbial fermentation in the ileum. Faecal looseness with the feeding of lactose is explicable.

Proteins

The amount of protein hydrolysed in the small intestine is about three times that in the stomach. Proteins are in the form of long folded chains, the links of which are represented by amino acid residues. For proteins to be digested and utilized by the horse these amino acids must usually be freed, although the gut mucosal cells can absorb dipeptides. The enzymes responsible are amino-peptidases and carboxy-peptidases secreted by the wall of the small intestine.

The loss of digesta from polyester bags passing from the stomach to the caecum, and containing either unmolassed sugar beet pulp, hay cubes, soya hulls or a 2:1 mixture of oat hulls:naked oats, has been measured (Moore-Colyer *et al.* (1997). The results (Table 1.2) indicate that beet pulp would be subject to greater hind-gut fermentation than would the other feeds.

Table 1.2 *In sacco* organic matter and crude protein (CP) disappearance from the DM or from the CP, in polyester bags during passage from the stomach to the caecum of Welsh cross ponies (Moore-Colyer *et al.* 1997).

	Component disappearance from the small intestine		
	Organic matter g/kg (DM)	Crude protein, g/kg (CP)	Digestible crude protein g/kg (DM)
Sugar beet pulp	185	296	30
Hay cubes	294	521	52
Soya hulls	239	597	60
Oat hulls:naked oats, 2:1	337	771	54

Fats

The horse differs from the ruminant in that the composition of its body fat is influenced by the composition of dietary fat. This suggests that fats are digested and absorbed from the small intestine before they can be altered by the bacteria of the large intestine. The small intestine is the primary site for the absorption of dietary fat and long-chain fatty acids. Bile continuously draining from the liver facilitates this by promoting emulsification of fat, chiefly through the agency of bile salts. The emulsification increases the fat–water interface so that the enzyme lipase may more readily hydrolyse neutral fats to fatty acids and glycerol. These are readily absorbed, although it is possible that a considerable proportion of dietary fat, as finely emulsified particles of neutral fat (triacylglycerols, TAG), is absorbed into the lymphatic system and transported as a lipoprotein in chylomicrons. Many research workers have demonstrated that horses digest fat quite efficiently and that the addition of edible fat to their diet has merit, particularly in so far as endurance work, and also more intensive exercise, are concerned (see Chapters 5 and 9).

Medium-chain TAG (carbon chain length of 6–12) are easily absorbed as such by horses, followed by portal transport to the liver, where they are metabolized to ketones (Jackson *et al.* 2001).

Feed modification to improve digestion

The extent of precaecal breakdown of cereal starch from pelleted diets is in the sequence: oat > barley > maize (de Fombelle *et al.* 2003). Varloud *et al.* (2003) and de Fombelle *et al.* (2003) found that although much starch disappeared (but was not absorbed) in the stomach, the amount escaping precaecal digestion increased with starch intake: 20% from barley and 30% from maize did so when horses received 281 g starch/100 kg BW in a meal. Thus, in order to increase digestibility and avoid fermentation of starch in the equine large gut, commercial cooking of cereals is of economic interest. The processes used include infrared micronization of cereals and expansion or extrusion of products. The extent of cooking by the extrusion process varies considerably amongst the cookers used and the conditions of processing. Nevertheless, small intestinal digestibility is influenced by this cooking, even in adult horses; yet total digestibility is not improved (Table 1.3). That is, the digestibility of raw cereals and cooked cereals is similar when the values are derived from the difference between carbohydrate consumed and that lost in the faeces. Thus, the extent of precaecal digestion, or possibly preileal digestion, influences the proportion of cereal carbohydrate absorbed as glucose and that absorbed as VFA and lactic acid.

Evidence from various sources indicates that somewhat more than 50% of the dietary starch is subject to preileal or precaecal digestion. The proportion so digested is influenced not only by cereal processing, but also by the amount fed. Lactate and other organic acid production is increased, and the pH is decreased in

Table 1.3 Precaecal digestion of various sources of starch and digestion in the total GI tract of horses (Kienzle *et al.* 1992) and ponies (Potter *et al.* 1992a) (digested, g/kg intake).

	Starch intake, g/100 kg BW	Maize precaecal	Oats precaecal	Oats total	Sorghum precaecal	Sorghum total	Reference
Whole	200	289	835	—	—	—	Kienzle <i>et al.</i> 1992 ¹
Rolled	200	299	852	—			Kienzle <i>et al.</i> 1992
Ground	200	706	980				Kienzle <i>et al.</i> 1992
Crimped ²	264 ^{CO} 295 ^{CS}	—	480	944	360	940	Potter <i>et al.</i> 1992a
Micronized	237 ^{MO} 283 ^{MS}	—	623	938	590	945	Potter <i>et al.</i> 1992a

¹Maize and oat digestibilities measured by these workers refer to preileal measurements.

²Dry rolled with corrugated rollers to crack the kernels. ^{CO}, crimped oats; ^{CS}, crimped sorghum; ^{MO}, micronized oats; ^{MS}, micronized sorghum.

the ileum and caecum when undigested starch reaches those regions. In order to avoid starch 'overload', and therefore excessive starch fermentation in the large intestine, Potter *et al.* (1992a) concluded that starch intake in horses, given two to three meals daily, should be limited to 4 g/kg BW per meal (also see Chapter 11). We consider that this limit is too liberal where there is risk of laminitis (see Laminitis, Chapter 11). The Texas group (Gibbs *et al.* 1996) have also found that when N intake is less than 125 mg/kg BW, 75–80% of the truly digestible protein of soya-bean meal is digested precaecally, and 20% is digested in the large intestine, while 10% is indigestible.

The preileal digestibility of oat starch exceeds that of maize starch or of barley starch (Meyer *et al.* 1995). When starch intake per meal is only 2 g/kg BW the preileal starch digestibility of ground oats may be over 95%, whereas at the other extreme that of whole, or broken, maize may be less than 30%. The grinding of cereals increases preileal digestibility compared with whole, rolled or cracked grain (note: the keeping quality, or shelf-life, of ground grain is, however, relatively short). Workers in Hanover found, in contrast to the results of the Texas workers, that in the jejunal chyme there is a much greater increase in the postprandial concentration of organic acids, including lactate, and in acidity, when oats rather than maize are fed. Whether this may be related to the putative heating effect of oats, compared with other cereals, is not established. The starch gelatinization of cooking enhances small intestinal digestion at moderate, or high, rates of intake.

Nitrogen utilization

At high rates of protein intake more non-protein N (NPN) enters the GI tract in the form of urea. The N entering the caecum from the ileum is proportionally 25–40%

NPN, varying with the feed type. Meyer (1983b) calculated that, in a 500 kg horse, 6–12 g urea N pass daily through the ileocaecal valve. The amount of N passing into the large intestine also varies with protein digestibility. At high intake rates of protein of low digestibility more N in total will flow into the large intestine, where it will be degraded to NH_3 . From Meyer's evidence, about 10–20% of this total is urea N, as the daily range of total N flowing into the caecum is:

$$0.3\text{--}0.9 \text{ g N/kg BW}^{0.75}$$

N also enters the large intestine by secretion there, although the amount seems to be less than that entering through the ileocaecal valve and net absorption nearly always takes place. Nevertheless, net secretion can occur with low protein, high fibre diets.

Utilization of the derived NH_3 by gut bacteria is between 80% and 100%. Excessive protein intake must increase the burden of unusable N, either in the form of inorganic N, or as relatively unusable bacterial protein. This burden is influenced by feeding sequence. The provision of a concentrate feed two hours later than roughage, compared with simultaneous feeding, caused higher levels of plasma free and particularly of essential amino acids six to nine hours later (Cabrera *et al.* 1992; Frape 1994). Plasma urea did not rise with the dissociated, or separate, feeding, but rose continuously for nine hours after the simultaneous feeding of the roughage and concentrate. This indicates that mixed feeding led to a large flow of digesta N to the caecum with much poorer dietary protein economy; yet the separate feeding was in the reverse order to the standard practice of giving concentrates before roughage.

THE LARGE INTESTINE

Grazing herbivores have a wide variety of mechanisms and anatomical arrangements for making use of the chemical energy locked up in the structural carbohydrates of plants. A characteristic of all grazing and browsing animals is the enlargement of some part of the GI tract to accommodate fermentation of digesta by microorganisms, producing steam VFAs and lactate (Table 1.4).

Table 1.4 Effect of diet on the pH, production of VFAs and lactate and on microbial growth in the caecum and ventral colon of the horse 7 hours after the meal.

Diet	pH	FA (mmol/l)				Total bacteria per (ml $\times 10^{-7}$)
		Acetate	Propionate	Butyrate	Lactate	
Hay	6.90	43	10	3	1	500
Concentrate plus minimal hay	6.25	54	15	5	21	800
Fasted	7.15	10	1	0.5	0.1	5

Note: Values given are typical, but all except the pH show large variations.

More than half the dry weight of faeces is bacteria and the bacterial cells in the digestive tract of the horse number more than ten times all the tissue cells in the body. No domestic mammal secretes enzymes capable of breaking down the complex molecules of cellulose, hemicellulose, pectin, fructo- and galacto oligosaccharides and lignin into their component parts suitable for absorption, but, with the exception of lignin, intestinal bacteria achieve this. The process is relatively slow in comparison to the digestion of starch and protein. This means that the flow of digesta has to be arrested for sufficient time to enable the process to reach a satisfactory conclusion from the point of view of the energy economy of the host animal.

During the weaning and postweaning of the foal and yearling, the large intestine grows faster than the remainder of the alimentary canal to accommodate a more fibrous and bulky diet, hence energy digestibility of a mixed concentrate and forage diet increases at 5–8 months of age (Turcott *et al.* 2003).

At the distal end of the ileum there is a large blind sack known as the caecum, which is about 1 m long in the adult horse and which has a capacity of 25–35 l. At one end there are two muscular valves in relatively close proximity to each other, one through which digesta enter from the ileum and the other through which passage from the caecum to the right ventral colon is facilitated. The right and left segments of the ventral colon and the left and right segments of the dorsal colon constitute the great colon, which is some 3–4 m long in the adult horse, having a capacity of more than double that of the caecum. The four parts of the great colon are connected by bends known as flexures. In sequence, these are the sternal, the pelvic and the diaphragmatic flexures (Fig. 1.2). Their significance probably lies in changes in function and microbial population from region to region and in acting as foci of intestinal impactions.

Digestion in the caecum and ventral colon depends almost entirely on the activity of their constituent bacteria and ciliate protozoa. In contrast to the small intestine, the walls of the large intestine contain only mucus-secreting glands, that is, they provide no digestive enzymes. However, high levels of alkaline phosphatase activity, known to be associated with high digestive and absorptive action, are found in the large intestine of the horse, unlike the large intestinal environment of the cat, dog and man.

The diameter of the great colon varies considerably from region to region but reaches a maximum in the right dorsal colon where it forms a large sacculum with a diameter of up to 500 mm. This structure is succeeded by a funnel-shaped part below the left kidney where the bore narrows to 70–100 mm as the digesta enter the small colon. The latter continues dorsally in the abdominal cavity for 3 m before the rectum, which is some 300 mm long, terminates in the anus (Fig. 1.2).

Contractions of the small and large intestine

The walls of the small and large intestine contain longitudinal and circular muscle fibres essential:

- for the contractions necessary in moving the digesta by the process of peristalsis in the ultimate direction of the anus;
- for allowing thorough admixture with digestive juices; and
- for bathing the absorptive surfaces of the wall with the products of digestion.

During abdominal pain these movements may stop so that the gases of fermentation accumulate.

Passage of digesta through the large intestine

Many digestive upsets are focused in the large intestine and therefore its function deserves discussion. The extent of intestinal contractions increases during feeding – large contractions of the caecum expel digesta into the ventral colon, but separate contractions expel gas, which is hurried through much of the colon. The reflux of digesta back into the caecum is largely prevented by the sigmoid configuration of the junction. Passage of digesta through the large intestine depends on gut motility, but is mainly a function of movement from one of the compartments to the next through a separating barrier. Considerable mixing occurs within each compartment, but there seems to be little retrograde flow between them. The barriers are:

- the ileocaecal valve already referred to;
- the caecoventral colonic valve;
- the ventrodorsal colonic flexure (pelvic flexure), which separates the ventral from the dorsal colon; and
- the dorsal small colonic junction at which the digesta enter the small colon.

Resistance to flow tends to increase in the same order, that is, the last of these barriers provides the greatest resistance (also see Chapter 11). This resistance is much greater for large food particles than for small particles. In fact delay in passage for particles of 2 cm length may be for more than a week. Normally the time taken for waste material to be voided after a meal is such that in ponies receiving a grain diet, 10% is voided after 24 hours, 50% after 36 hours and 95% after 65 hours. More recently, mean retention time (MRT) in 18-month-old horses given a hay and concentrate diet was shown to be 42.7 and 33.8 hours respectively for the solid and liquid phases of digesta (Chiara *et al.* 2003), and for a hay-based diet in mature heavy horses it was 21–40 hours, decreasing within this range as intake increased (Miraglia *et al.* 2003). Within moderate variations of intake the digestibility of the diet was constant. A large decrease in MRT was associated with a lower digestibility coefficient.

Most digesta reach the caecum and ventral colon within three hours of a meal, so that it is in the large intestine that unabsorbed material spends the greater proportion of time. The rate of passage in domestic ruminants is somewhat slower, and this partly explains their greater efficiency in digesting fibre. Nevertheless, the horse, utilizes the energy of soluble carbohydrates more efficiently by absorbing a greater proportion of sugars in the small intestine.

In the horse, passage time is influenced by physical form of the diet; for example, pelleted diets have a faster rate of passage than chopped or long hay, and fresh grass moves more rapidly than hay. Work at Edinburgh (Cuddeford *et al.* 1992) showed that fibre was digested more completely by the donkey than by the pony, which in turn digested it more effectively than the TB. These differences probably are owed in large measure to the relative sizes of the hind-gut and, therefore, to the holding time of digesta. Donkeys working for five hours daily with no access to food, subsequently ate as much poor quality hay and digested it as well as those not working and with continuous access (Nengomasha *et al.* 1999). Holding time in the large gut seems to be uninfluenced by meal size, whereas rate of passage through the small intestine is greater with less frequent large meals.

Pattern of large intestinal contractions

The caecum contracts in a ring some 12–15 cm from the caecocolic junction, trapping ingesta in the caecal base and forcing some through the junction that in the meantime has relaxed. With a relaxation of the caecal muscles some reflux occurs, although there is a net movement of digesta into the ventral colon. The passage rate of digesta through the caecum is approximately 20%/hour (Hintz 1990), compared with a typical rate for the rumen of 2–8%/hour. However, disappearance rates of feed in monofilament polyester bags held in the pony caecum were greater during hay feeding than between meals (Hyslop *et al.* 1999). Feeding seems to cause an increase in the motility and volume of the caecum, allowing a more thorough mixing of its digesta with the bacteria.

Contractions of the colon are complex. There are bursts of contractile activity that propagate in an aboral direction, but some contractions propagate orally and some are isolated and do not propagate in either direction. Thus there are nonrhythmic haustral kneading and stronger rhythmic propulsive and retropulsive contractions. These contractions have the function of mixing the constituents, and promoting fermentation and absorption, as well as that of moving residues towards the rectum. The strong rhythmic contractions for the great colon begin at the pelvic flexure, where a variable site ‘electrical pacemaker’ exists. A major site of impactions is the left ventral colon, just orad (toward the mouth) to this pelvic flexure (Chapter 11). More detailed knowledge of this activity should ultimately help in the control of common causes of large gut malfunction and colic.

Microbial digestion (fermentation)

There are three main distinctions between microbial fermentation of feed and digestion brought about by the horse’s own secretions:

- (1) The β -1,4-linked polymers of cellulose (Fig. 1.3) are degraded by the intestinal microflora but not by the horse’s own secretions. The cell walls of plants

contain several carbohydrates (including hemicellulose) that form up to half the fibre of the cell walls of grasses and a quarter of those of clover. These carbohydrates are also digested by microorganisms, but the extent depends on the structure and degree of encrustation with lignin, which is indigestible to both gut bacteria and horse secretions (see 'Flora', this chapter).

- (2) During their growth the microorganisms synthesize dietary indispensable (essential) amino acids.
- (3) The bacteria are net producers of water-soluble vitamins of the B group and of vitamin K₂.

Microbial numbers

In the relatively small fundic region of the stomach, where the pH is about 5.4, there are normally from 10^8 to 10^9 bacteria/g. The species present are those that can withstand moderate acidity, common types being lactobacilli, streptococci and *Veillonella gazogenes*. De Fombelle *et al.* (2003) found that lactobacilli-, streptococci- and lactate-utilizing bacteria colonized the entire GI tract. The stomach and small intestine hosted, per ml, the greatest number of these bacteria, so influencing the digestion of readily fermentable carbohydrates. De Fombelle *et al.* also determined that the highest concentration of total anaerobic bacteria in the GI tract occurred in the stomach (see Gastric ulcers, Chapter 11). The jejunum and ileum support a flourishing population in which obligate anaerobic Gram-positive bacteria may predominate (10^8 – 10^9 /g). In this region of the small intestine a cereal diet can influence the proportion of the population producing lactic acid, compared with that producing VFA as an end product, although the numbers of lactobacilli per gram of contents tend to be higher in the large intestine, where the pH is generally lower.

The flora of the caecum and colon are mainly bacteria which in fed animals number about 0.5×10^9 to 5×10^9 /g contents. A characteristic difference between equine hind-gut fermentation and that in the rumen is the lower starch content of the hind-gut, which implies a generally lower rate of fermentation, yet the starch content of the caecum is variable, causing a variable suppression of cellulolytic and related bacteria. As the proportion of rolled barley to chopped meadow hay (given after the barley) was increased from nil to half, the digestibility of OM increased, whereas that of neutral detergent fibre (NDF) and acid detergent fibre (ADF) decreased, despite the retarded flow rate of digesta with the higher proportions of barley (C. Drogoul, personal communication).

There is still a scarcity of knowledge concerning the activity of equine bacteria that digest the various entities of fibre. In one pony study (Moore & Dehority 1993), the cellulolytic bacteria numbered 2–4% of the total. In addition, there were 2×10^2 to 25×10^2 fungal units/g, most of which were cellulolytic (also see 'Probiotics', Chapter 5). In the horse, both caecal bacteria (which with fungi constitute the flora) and protozoa (fauna) participate in the decomposition of pectins and hemicellulose at an optimum pH of 5–6 (Bonhomme-Florentin 1988).

Fauna

Protozoa in the equine large intestine number about 10^{-4} of the bacterial population, that is, 0.5×10^5 to 1.5×10^5 /ml contents. Although protozoa are individually very much larger than bacteria and they thus contribute a similar total mass to the large intestinal contents, their contribution to metabolism is less, as this is roughly proportional to the surface area. The species of the fauna differ somewhat from those in the rumen. Some 72 species of protozoa, primarily ciliates, have been described as normal inhabitants of the equine large intestine, with some tendency to species differences between compartments. Moore & Dehority (1993) found in ponies that the protozoa were from the following genera: *Buetschlia*, *Cycloposthium*, *Blepharocorys* and a few *Paraisotricha*. Removal of the protozoa (defaunation) caused only a slight decrease in DM digestibility, with no effect on numbers of bacteria, or on cellulose digestibility.

Flora

In the large intestine the bacterial populations are highest in the caecum and ventral colon. Here, the concentration of cellulose-digesting bacteria is six to seven times higher than in the terminal colon. About 20% of the bacteria in the large intestine can degrade protein.

Numbers of specific microorganisms may change by more than 100-fold during 24 hours in domesticated horses being given, say, two discrete meals per day. These fluctuations reflect changes in the availability of nutrients (in particular, starch and protein) and consequentially changes in the pH of the medium. Thus, a change in the dietary ratio of cereal to hay will not only have large effects on the numbers of microorganisms but will also considerably influence the species distribution in the hindgut. Although frequency of feeding may have little impact on digestibility per se, it can have a large influence on the incidence of digestive disorders and metabolic upsets. Large concentrate meals lead to elevated glycaemic responses that can precipitate behavioural abnormalities, whereas fibrous feed lowers this response. Moreover, fibre stimulates peristalsis and is cationic, decreasing the risk of metabolic acidosis (Moore-Colyer 1998). Some of these consequences result directly from the effects of diet and digesta upon the microbial populations (bacteria and protozoa).

Caecal bacteria from horses adapted to a grain diet are less efficient at digesting hay than are the microbes from hay-adapted horses. An analogous situation exists for hay-adapted caecal microbes when subjected to grain substrate. If such a dietary change is made abruptly in the horse, impactions may occur in the first of these situations and colic, laminitis or puffy swollen legs can result in the second (see Chapter 11).

The caecal microorganisms in a pony or horse tend to be less efficient at digesting hay than are the ruminal microbes in cows. The digestibilities of organic matter and crude fibre in horses given a diet containing more than 15% crude fibre (a normal

diet of concentrates and hay) are about 85% and 70–75%, respectively, of the ruminant values. This has been attributed to the combined effects of a more rapid rate of passage of residues in horses and differences in cellulolytic microbial species. In fact, Hayes *et al.* (2003) concluded that a greater intestinal retention time in mares cf. foals of one month of age, accounted for their greater ability to digest fibre. The faecal population of microbes from these foals had a capacity similar to that of the mare to ferment fescue hay NDF. Differences also occur amongst regions of the GI tract in the time required for microbial enzyme adaptation to fibre fermentation. This will influence the extent of fermentation in a limited time. Inocula from the stomach, duodenum and ileum expressed a lag time of 1–2 hours, cf. 0.1–0.5 hours for hind-gut inocula, in roughage fermentation, so limiting foregut fermentation (Moore-Colyer *et al.* 2003).

Work by Hyslop *et al.* (1997) has shown that under the conditions of their experiment the degradation of the acid detergent fibre (ADF) and crude protein of sugar beet pulp, hay cubes, soya hulls and a 2:1 mixture of oat hulls:naked oats was no poorer in the pony caecum than in the rumen of the steer over incubation periods of 12–48 hours. In fact, during incubation for 12 hours the degradation of the beet pulp and the hay was marginally greater in the caecum. Thus, the equine hind-gut microflora may not be inherently less efficient than are rumen microflora at feed degradation. Lower equine feed digestibility largely results from a more rapid rate of passage through the hind-gut than through the rumen (Hyslop *et al.* 1997).

Estimation of fibre degradability

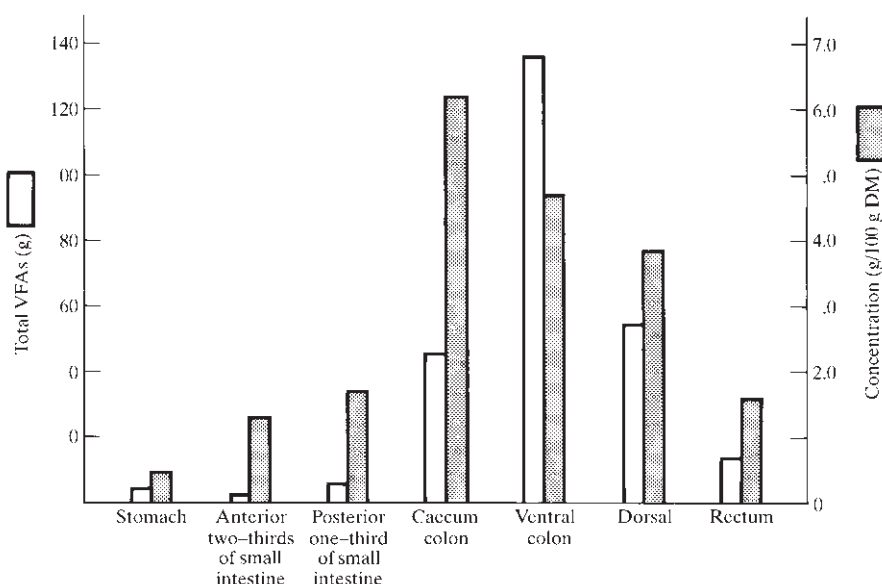
Moore-Colyer (1998) measured apparent digestibility and fibre degradation, as indicated by analysis of non-starch polysaccharides (NSPs) and NDF, of sugar beet pulp (SB), soya hulls (SHs), hay cubes (HCs) and oat hulls:naked oats (OH:NO) (2:1). NSP molecules are composed of several constituent monomers that are present in different proportions in various sources, and these monomers are normal components of cell walls. The principal monomers are: arabinose, galactose, uronic acids, glucose and xylose. The most *microbially* degradable monomers in the above four feeds were arabinose, galactose and uronic acids. SB had the highest concentrations of arabinose and uronic acids and was degraded at the fastest rate, whereas the rates for HC and particularly for OH were much slower. HC and OH would have a lower apparent digestibility than SB, and SH would be intermediate in value. NSP and NDF are simpler to measure, but are poorer guides to degradability than is knowledge of the monomer composition of the NSP of feeds. The subject of fibre analysis has been reviewed recently in several papers, notably by McCleary (2003).

Products of fermentation

The microbial fermentation of dietary fibre, starch and protein yields large quantities of short-chain VFAs as by-products, principally acetic, propionic and butyric acids (Table 1.5, Fig. 1.4). This fermentation and VFA absorption are promoted by:

Table 1.5 Proportion of VFAs in digesta to body weight (BW) in four herbivores (Elsden *et al.* 1946).

	g VFA/kg BW
Ox	1.5
Sheep	1.5
Horse	1.0
Rabbit	0.5

**Fig. 1.4** VFAs (□) calculated as the total weight (g) of acid (as acetic acid) in the organ or as the concentration (g/100 g DM) (■) in the lumen (after Elsden *et al.* 1946).

- the buffering effect of bicarbonate and Na^+ derived from the ileum;
- an anaerobic environment; and
- normal motility to ensure adequate fermentation time and mixing.

Acetate and butyrate are major products of fibre digestion, whereas the proportion of propionate (and lactate, see Chapter 11) increases with increasing proportions of starch left undigested in the small intestine. In the pony, limited evidence indicates that 7% of total glucose production is derived from propionate produced in the caecum.

VFA, fluid and electrolyte absorption in the large intestine

The VFA produced during fermentation would soon pollute the medium, rapidly producing an environment unsuitable for continued microbial growth; however, an

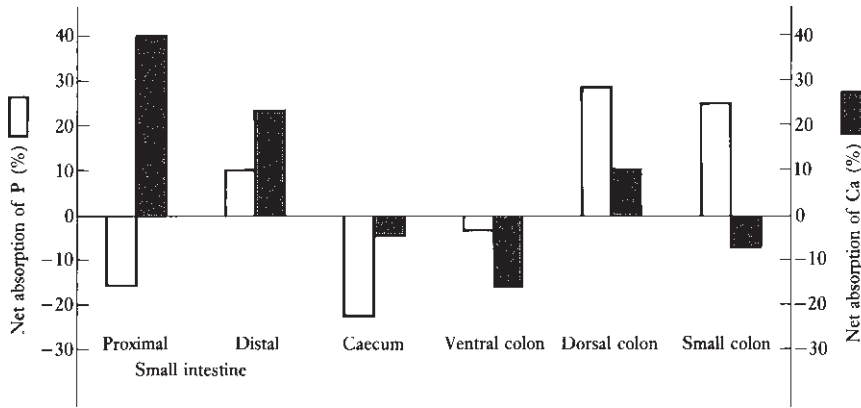


Fig. 1.5 Net fractional absorption of P (□) and Ca (■) from various regions of the small and large intestine (after Schryver *et al.* 1974a).

equable medium is maintained by the absorption of these acids into the bloodstream. In addition to this absorption there is the vital absorption of large amounts of water and electrolytes (sodium, potassium, chloride and phosphate).

Fluid absorption

The largest proportion of water that moves through the ileocaecal junction is absorbed from the lumen of the caecum and the next largest is absorbed from the ventral colon. Fluid is also absorbed from the contents of the small colon, to the benefit of the water economy of the horse and with the formation of faecal balls. This aboral decline in water absorption is accompanied by a parallel decrease in sodium absorption. In the pony, 96% of the sodium and chloride and 75% of the soluble potassium and phosphate entering the large bowel from the ileum are absorbed into the bloodstream. Although phosphate is efficiently absorbed from both the small and large gut, calcium and magnesium are not, these being absorbed mainly from the small intestine (Fig. 1.5). This phenomenon has been proffered as a reason why excess dietary calcium does not depress phosphate absorption, but excess phosphate can depress calcium absorption, although not necessarily Ca balance, in the horse (see Chapter 3).

The water content of the small intestinal digesta amounts to some 87–93%, but the faeces of healthy horses contain only 58–62% water. The type of diet has a smaller effect on this than might be imagined. For instance, oats produce fairly dry faeces, but bran produces moist faeces, although in fact they contain only some 2 or 3 percentage units more of moisture.

VFA and lactic acid production and absorption

Microbial degradation seems to occur at a far faster rate in the caecum and ventral colon than in the dorsal colon (Fig. 1.4) and the rate is also faster when starches are degraded rather than structural carbohydrates. A change in the ratio of starch to

fibre in the diet leads to a change in the proportions of the various acids yielded (Table 1.4). These proportions also differ in the organs of the large intestine. Thus, proportionately more propionate is produced as a consequence of the consumption of a starch diet and the caecum and ventral colon yield more propionate than the dorsal colon does. Many bacteria have the capacity to degrade dietary protein, so yielding another blend of VFA.

An optimum pH of 6.5 exists for microbial activity that also promotes VFA absorption. VFAs are absorbed in the unionized form. As the pH moves closer to the pK of a particular VFA, more is absorbed. The H^+ ions required for this are probably derived from mucosal cells in exchange for Na^+ . HCO_3^- buffer is secreted into the lumen in exchange for Cl^- . Thus, absorption of VFAs is accompanied by a net absorption of NaCl. This in turn is a major determinant of water absorption. The ingestion of a large meal can cause a 15% reduction in plasma volume, ultimately resulting in renin–angiotensin, and then aldosterone, release. The increase in plasma aldosterone level causes an increased Na^+ absorption, and with it water (see also Chapter 9). However, whether a large meal, compared with continuous feeding, would increase the risk of impactions is unclear.

Whereas most ruminal butyrate is metabolized in the mucosa before entering the bloodstream, in horses all VFAs pass readily to the blood. Lactic acid produced in the stomach is apparently not well absorbed from the small intestine. On reaching the large intestine some is absorbed, along with that produced locally, but much is metabolized by bacteria to propionate.

Microbial activity inevitably produces gases – principally carbon dioxide, methane and small amounts of hydrogen – which are absorbed, ejected from the anus, or participate in further metabolism. The gases can, however, be a severe burden, with critical consequences when production rate exceeds that of disposal.

Protein degradation in the large intestine and amino acid absorption

Microbial growth, and therefore the breakdown of dietary fibre, also depends on a readily available source of nitrogen. This is supplied as dietary proteins and as urea secreted into the lumen from the blood. Despite the proteolytic activity of microorganisms in the hind-gut, protein breakdown per litre is about 40-fold greater in the ileum than in the caecum or colon, through the activity of the horse's own digestive secretions in the small intestine.

The death and breakdown of microorganisms within the large intestine release proteins and amino acids. The extent to which nitrogen is absorbed from the large intestine in the form of amino acids and peptides useful to the host is still debated. Isotope studies indicate that microbial amino acid synthesis within the hind-gut does not play a significant role in the host's amino acid status. Quantitative estimates obviously depend on the diet used and animal requirements, but a range of 1–12% of plasma amino acids may be of hind-gut microbial origin. Absorption studies have shown that, whereas ammonia is readily absorbed by the proximal colon, significant basic amino acid absorption does not occur. S-amino acid absorption may occur to a small extent. Consequently, small-intestinal digestibility of protein is important,

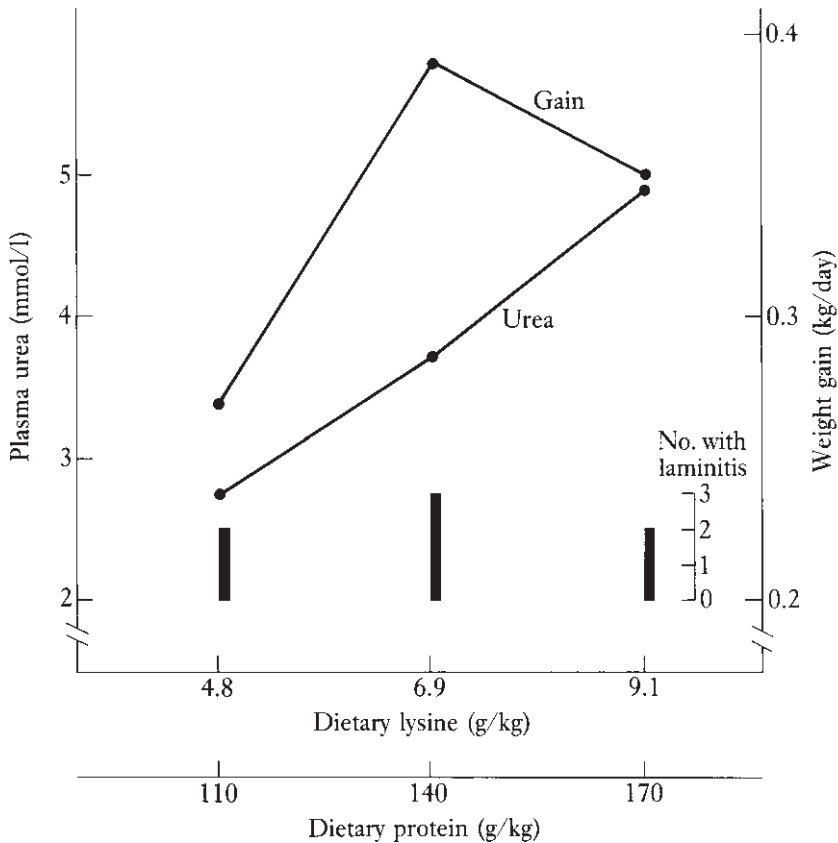


Fig. 1.6 Response of 41 six-month-old ponies over a three-month period of diets containing different amounts of protein and lysine (initial weight 127 kg). In this experiment note that increased protein intake led to elevated protein catabolism and urea production without an increase in incidence of laminitis (Yoakam *et al.* 1978).

and this digestibility of sugar-beet pulp is somewhat poorer than that of hay cubes and much poorer than that of soya hulls (Moore-Colyer 1998). The latter, therefore, possess the highest amino acid value of the three.

Horses differ from ruminants in absorbing a higher proportion of dietary nitrogen in the form of the amino acids present in dietary proteins, proportionately less being converted to microbial protein. As only a small proportion of the amino acids present in microbial protein is made available for direct utilization by the horse, young growing horses in particular respond to supplementation of poor-quality dietary protein with lysine and threonine, the principal limiting indispensable amino acids (Fig. 1.6).

Urea production

Urea is a principal end-product of protein catabolism in mammals and much of it is excreted through the kidneys. It is a highly soluble, relatively innocuous compound

Table 1.6 Effect of diet on the flow of nitrogen from the ileum to the caecum (Schmidt *et al.* 1982).

Diet	Nitrogen flow daily (mg N/kg BW)
Concentrate, 3.75 kg daily (1%)*	62
Concentrate, 7.5 kg daily (2%)*	113
Hay	68
Straw	37

* Weight of concentrate given as percentage of BW.

and a reasonably high proportion of the urea produced in the liver is secreted into the ileum and conveyed to the large intestine (Table 1.6, showing total N, of which 12–24 mg/kg BW is urea N) where most may be degraded to ammonia by bacteria. The possession by microorganisms of the enzyme urease, which does not occur in mammalian cells, makes this reaction possible. Most of the ammonia produced is re-utilized by the intestinal bacteria in protein synthesis. Some, however, diffuses into the blood, where levels are normally maintained very low by a healthy liver. If ammonia production greatly exceeds the capacity of the bacteria, and of the liver, to utilize it, ammonia toxicity can arise. The fate of any urea added to the diet is similar.

In summary, many studies have led to the conclusion that digestion and fermentation in, and absorption from, the large intestine, account, in net terms, for 30% of dietary protein, 15–30% of dietary soluble carbohydrate and 75–85% of dietary structural carbohydrate. The salient causes of variation in values for each of the principal components of the horse's diet are:

- the degree of adaptation of the animal;
- the processing to which the feed is subjected; and
- the differences in digestibility among alternative feedstuffs.

Commercial enzyme and microorganism products

(See also 'Probiotics', Chapter 5.) A Directive from the European Union (EU) (Council Directives 93/113/EC 93/114/EC) deals with the roles of supplementary enzymes and microbial cultures. There is a list of all permitted and properly identified products for use within the EU. The list includes products for use in feed, in drinking water and those given as a drench. The requirement for a complete taxonomic description of microorganisms implies that bacterial cultures should be pure and that there is an identification of both the species and the culture collection strain type. The recommendations of the Nomenclature Committee of the International Union of Biochemistry and Molecular Biology are applied to enzyme nomenclature. The United Kingdom (UK) Medicines Act (1986) will apply if medicinal

claims, such as growth promotion, are made; however, evidence of safety of all products will have been submitted.

Efficacy of products such as these may depend on conditions and length of storage, the loss of colony-forming units of microbial cultures during storage, their ability to survive the low pH of the stomach and certainly the retention of the activity of their enzymes. The efficacy of enzymes also must assume that their activity, as defined, applies to the region of the GI tract where they are expected to function.

STUDY QUESTIONS

- (1) What are the advantages and disadvantages of a digestive system with a major microbial fermentation site in the hind-gut only, compared to the fermentation system of the ruminant?
- (2) The stomach of the horse is relatively smaller than that of the rat or human. What consequences do you draw from this?

FURTHER READING

- Sissons, S. & Grossman, J.D. (1961) *The Anatomy of the Domestic Animals*. W.B. Saunders, Philadelphia and London.
- Vernet, J., Vermorel, M. & Martin-Rosset, W. (1995) Energy cost of eating long hay, straw and pelleted food in sport horses. *Animal Science*, **61**, 581–8.

Chapter 2

Utilization of the Products of Dietary Energy and Protein

A horse whose work consists of travelling a stage of twenty miles three times a week, or twelve every day, should have one peck of good oats, and never more than eight pounds of good hay in twenty-four hours. The hay, as well as the corn, should, if possible, be divided into four portions.

J. White 1823

CARBOHYDRATE, FAT AND PROTEIN AS SOURCES OF ENERGY, AND THE HORMONAL REGULATION OF ENERGY

Glucose, VFA and TAG clearance

Horse diets rarely contain more than 5% fat and 7–12% protein so that these represent relatively minor sources of energy in comparison to carbohydrate, which may constitute by weight two-thirds of the diet. Furthermore, protein is required primarily in the building and replacement of tissues and is an expensive source of energy. However, both dietary protein and fat can contribute to those substrates used by the horse to meet its energy demands for work. Protein does so by the conversion of the carbon chain of amino acids to intermediary acids and of some of the carbon chains to glucose. Neutral fat does so following its hydrolysis to glycerol and fatty acids. The glycerol can be converted to glucose and the fatty acid chain can be broken down by a stepwise process called β oxidation in the mitochondria, yielding adenosine triphosphate (ATP) and acetate, or, more strictly, acetyl coenzyme A (acetyl-CoA), and requiring tissue oxygen (see Fig. 9.2, p. 307).

Carbohydrate digestion and fermentation yield predominantly glucose and acetic, propionic and butyric VFAs. These nutrients are collected by the portal venous system draining the intestine and a proportion of them is removed from the blood as they pass through the liver. Both glucose and propionate contribute to liver starch (glycogen) reserves, and acetate and butyrate bolster the fat pool (Fig. 2.1) and also constitute primary energy sources for many tissues.

Sequence of feeding and amount fed

Studies in both France and Germany have shown that the sequence in which feeds are given can influence the metabolic outcome. When the concentrate was given to ponies two hours following roughage, plasma urea concentration was significantly

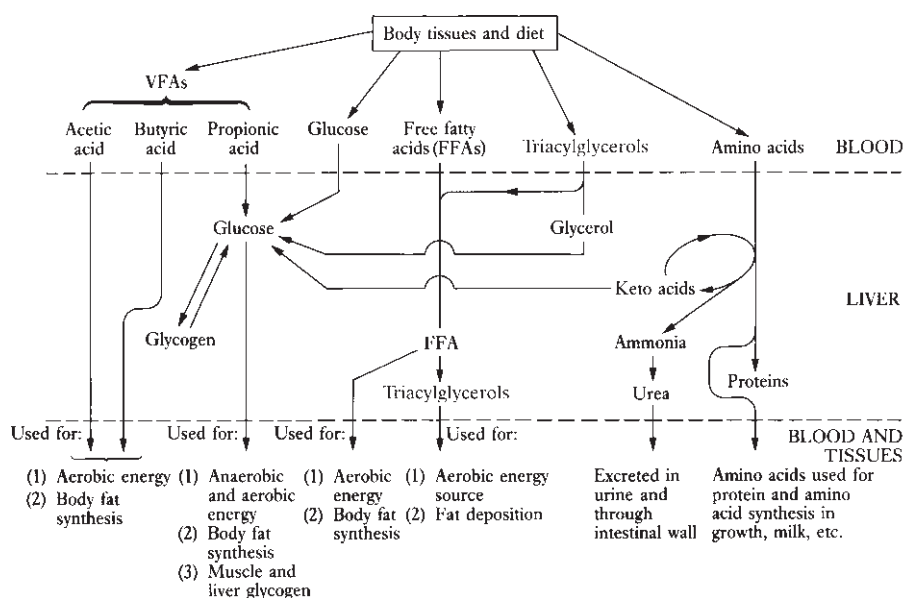


Fig. 2.1 Sources, metabolism and fate of major energy-yielding nutrients derived from body tissues and diet (after McDonald *et al.* 1981).

lower, and plasma free amino acids higher, in the postprandial period, than when the concentrates and roughage were given simultaneously (Cabrera *et al.* 1992). This indicates that the conventional procedure of feeding the concentrate with, or before, the hay is likely to depress the potential net protein value of the diet. Concentrates appear to be better utilized if given after roughage consumption, probably as a result of retarded small intestinal passage of the concentrates so given. Larger feed allowances increase the rate of passage of ingesta through the GI tract, as occurs for horses commencing periods of greater work. Increased rates of passage slightly reduce roughage digestibility and account for somewhat poorer fibre utilization in ponies than in donkeys (Pearson & Merritt 1991).

Timing of feeds and appetite

(See 'Appetite', this chapter, for more detail.) Feeding in the horse or pony causes mesenteric hyperaemia, that is, a diversion of blood to, and engorgement of, the blood vessels investing the GI tract. In a similar manner the imposition of exercise causes increased blood flow to muscles. Exercise within a few hours of feeding therefore increases the demands on the heart to supply blood for both activities. Even moderately strenuous exercise (75% of heart rate maximum) under these conditions leads to increased heart rate, cardiac output, stroke volume and arterial pressure, in comparison to the effects of exercise on fasted animals (Duren *et al.* 1992). The optimum timing of meals in relation to exercise is discussed in Chapter 9.

Blood glucose

Healthy horses and ponies maintain a blood plasma glucose concentration within certain defined limits. This is necessary as glucose represents the preferred source of energy for most tissues. In ponies, normal healthy resting levels may range between 2.8 and 3.3 mmol/l, but horse breeds may generally have higher resting levels, with TBs in the region of 4.4–4.7 mmol/l. The concentration in horses rises dramatically from the commencement of a meal to 6.5 mmol/l, or more, by two hours (Fig. 2.2). A return to fasting concentrations is much slower than in the human and slower still in ponies. Nevertheless, the scale of plasma glucose response to a meal is greatly influenced by the intensity of any previous exercise, intense exercise greatly diminishing the response (Frape 1989).

The plasma glucose response is measured generally as the area under the response curve to a known dose of carbohydrate. The more rapidly the plasma glucose is cleared, that is, the greater the tolerance, the smaller is this area (Figs 2.2 and 2.3). This clearance from the blood results from uptake particularly by liver and muscle cells, where it is converted to glycogen and also to fat, although the net conversion to fat in a fit athletic animal is small. Circulating glucose is used directly to meet immediate energy demands for muscular activity, nervous tissue activity, etc. The

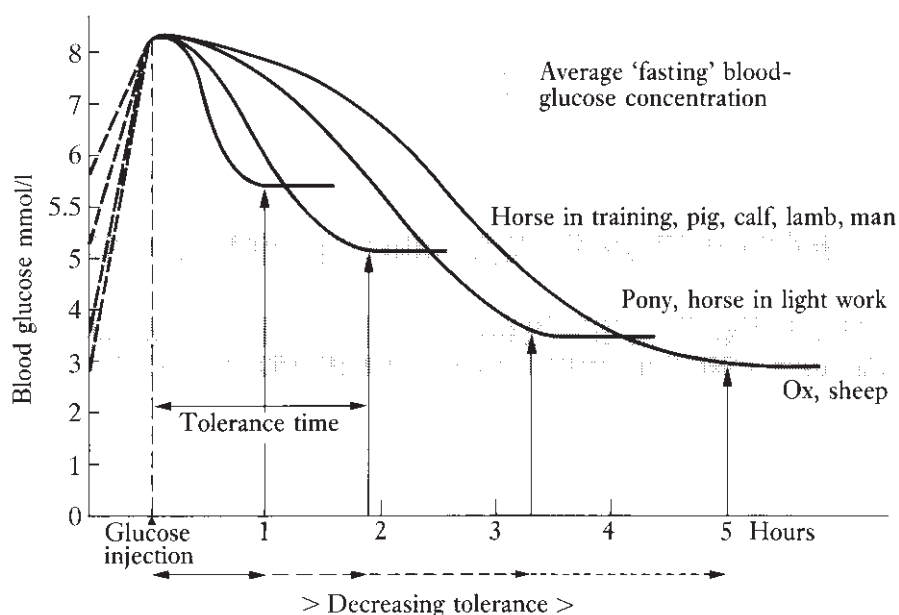


Fig. 2.2 Approximate glucose tolerance times (arrows) and normal 'fasting' blood-glucose concentration (shaded). (Glucose is injected i.v. to allow comparisons between species with different digestive anatomy and mechanisms. By providing the glucose in the form of a starch meal, the peak is delayed 2–4 hours in the horse. When oats are given as feed the maximum blood-glucose concentration in TB horses occurs at about 2 hours following the start of feeding.) The determination of 'tolerance time' has been largely superseded by the determination of the area under the response curve to a glucose dose, as this is determined with greater precision. The greater the area is, generally the longer is the tolerance time in an individual horse.

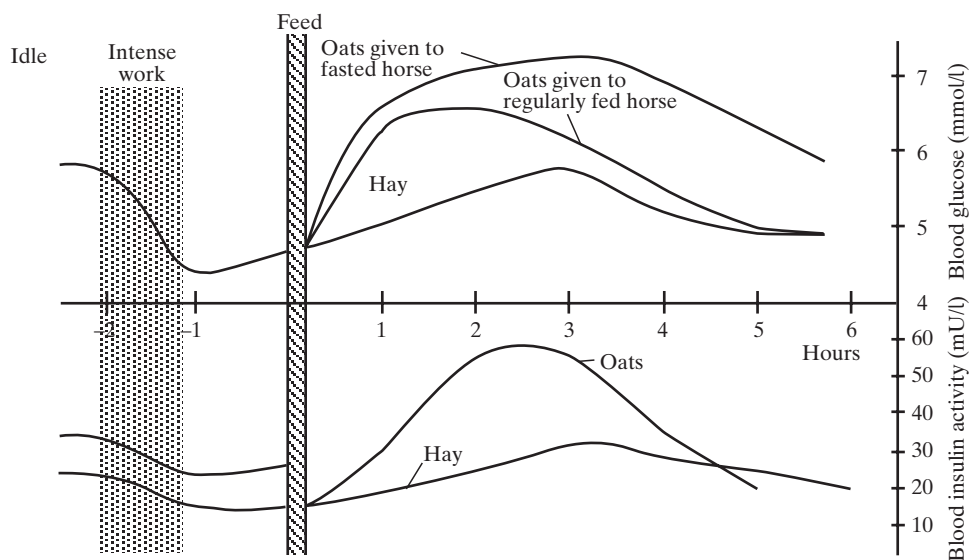


Fig. 2.3 Responses of blood glucose and insulin to feed.

process of storage is stimulated by the hormone insulin, which responds to a rise in blood glucose, and the insulin then also promotes fat clearance through activation of adipose tissue lipoprotein lipase.

Concomitant with the rise in plasma glucose concentration, the plasma non-esterified fatty acids (NEFAs) level decreases. This indicates a reduction in the mobilization of fat stores, which results from insulin repressing the activity of intracellular, hormone-sensitive lipase (also see Chapter 9). Generally, plasma glucose and insulin concentrations are lower and plasma NEFA and urea concentrations higher in horses given very restricted rations. Stickler *et al.* (1996) found that these plasma changes occurred promptly in light horses, whereas plasma glucagon responds with a slightly slower rate of decrease and thyroxine with a much slower increase in concentration upon the imposition of the restriction.

Pathologically elevated fasting plasma glucose concentrations occur in hyperadrenocorticism, a hypersecretion of cortisol, or Cushing's syndrome. This is associated with *luxus gluconeogenesis*, severe muscle wasting and protein breakdown. The resulting excessive production of glucose in the horse is due most frequently to the presence of a pituitary adenoma, although primary adrenocortical neoplasms have been reported (van der Kolk *et al.* 2001) (see also 'Amino acids', this chapter).

Insulin response

Insulin is an anabolic hormone, the function of which is therefore primarily to switch on enzymes directed towards storage of blood glucose and fat. Blood insulin peaks shortly after that of blood glucose and concentrations may reach four- to eightfold fasting values one to two hours following a feed. Blood insulin may remain above

fasting concentrations throughout the day, again unlike the human response to a single meal (Frape 1989). Horses and ponies have a lower glucose tolerance than do man and the pig, but a slightly greater one than do ruminants. TB and other hotblooded horses generally have a higher tolerance than do ponies; in other words, ponies tend to secrete less insulin and their tissues may be less sensitive to insulin, although there is considerable adaptation to diet. Ponies can therefore withstand fasting better than TBs, and TBs become more excitable after feeding.

Insulin prevents excess blood glucose from spilling out in the urine by increasing the uptake of tissues and so lowering blood levels. However, in order to avoid hypoglycaemia its effects are counterbalanced by that of other hormones (for example, glucagon, the glucocorticoids and the catecholamines, epinephrine and nor-epinephrine). The system is thereby maintained in a state of dynamic equilibrium. The anabolic nature of insulin conflicts with the catabolic requirements of exercise, and although catecholamines, secreted during intense exercise, suppress further insulin secretion, elevated circulating postprandial insulin is undesirable if exercise is initiated. Both from this viewpoint and from that of blood redistribution exercise is to be discouraged during the postprandial interval.

Cases of hypoglycaemic seizures have been reported in horses, during which plasma glucose may fall to less than 2 mmol/l. The cause has been adenoma of pancreatic islet cell origin, with hyperplasia predominately of β -cells, causing hyperinsulinaemia.

Insulin resistance and hyperinsulinaemia

Insulin-dependent diabetes is very rare in horses; however, the noninsulin-dependent form, expressed as insulin resistance, does occur. Whether any of these occurrences involve a deficiency of dietary trivalent chromium in horses is unknown (see Chapter 3).

Resistance may sometimes be incorrectly inferred. Horses given a diet high in starch content produced a similar glucose response to that of horses given a diet containing less starch, but the insulin response was higher in the first group (Ralston 1992). This may not indicate that there is a greater risk of developing insulin resistance for horses given high cereal diets. Insulin resistance is associated with an inadequate response of tissue receptors to the hormone; that is, a higher than normal local concentration of the hormone is required to elicit a normal tissue response. Plasma glucose and insulin responses to a meal, or to an oral glucose dose, are above normal. Ponies tend to be more intolerant of glucose than are horses, and fasted animals are more intolerant than fed. However, the response of ponies, and of Shetland ponies in particular, is a *reduced* insulin secretion in response to a carbohydrate load. This does not imply insulin resistance and it would explain a lesser decrease in plasma NEFA concentration in ponies following a glucose load. Oral glucose tolerance tests conducted on foal and adult ponies, given a pelleted high fibre diet, indicated that the adults were more glucose tolerant than either the foals, or adults given long hay (Murphy *et al.* 1997). (The oral glucose tolerance test

may suffer from the influence of factors other than insulin, for instance, impaired gut function, and the intravenous loading route may be preferable; see Chapter 12.)

Plasma TAG concentration in ponies subjected to an extended fast is very much higher than that in horses; although this may not indicate insulin resistance. It may result from the raised plasma NEFAs that are converted by the liver to TAGs, where they are mobilized and transported as very low density lipoproteins. (Glucose- and fat [TAG]-sensitive insulin tissue receptors exist in adipocytes. Insulin resistance retards fat deposition, and raised plasma TAG promotes insulin resistance, so it may not be coincidental that hyperlipaemia occurs more frequently in ponies than in horses.)

Dietary causes of insulin resistance remain unresolved. The principal dietary and metabolic involvement in the causation of resistance is likely to be excessive feed energy intake, obesity, ageing and inadequate exercise. Ponies previously suffering laminitis are also much more glucose intolerant (Jeffcott *et al.* 1986) than are those that have not had laminitis. Obesity in humans delays the plasma clearance of fat from a meal, owing to low insulin sensitivity of receptors in adipocytes. It has been shown that four days of fasting can cause visibly lipaemic plasma in ponies but not in horses, and the same factors may be a cause of laminitis. Moderate regular exercise may prevent both laminitis and insulin resistance. In humans, raised post-prandial plasma fat, following a fatty meal, causes a rise in plasma NEFA, which in turn may promote insulin resistance. Whether hyperlipaemia in ponies is a cause of insulin resistance, through an elevation in plasma NEFA in nonexercising ponies, has not been examined, but deserves to be so. Such a rise is unlikely to occur after a meal, as normal feeds are low in fat content.

ENERGY METABOLISM

Hard muscular work may require that energy is available for muscular contraction at a rate some 40 times that needed for normal resting activity. Thus, rapid changes in the supply of blood glucose could result unless the animal's system responds quickly. There are many changes to accommodate the altered circumstances, but our discussion at this point will relate to the supply of nutrients to the tissues.

During a gallop, pulmonary ventilation increases rapidly so that more oxygen (O_2) is available for transport by the blood to the skeletal and cardiac muscles for the oxidative release of energy. However, this process cannot keep pace with the demand for energy, and glucose is therefore broken down to lactic acid, rapidly releasing energy in the absence of O_2 . The fall in blood glucose stimulates the glucocorticoids and the other hormones that enhance glycogen breakdown so that blood glucose can rise during moderate exercise.

Repeated hard work ('Training', see Chapter 9) brings about several useful physiological adaptations to meet the energy demands of muscular work. First, the pulmonary volume, and therefore the tidal volume, of O_2 increases and the diffusion capacity for gases increases, so that carbon dioxide (CO_2) is disposed of more

efficiently from the blood and O_2 is absorbed at a faster rate. This process is greatly assisted by changes in both numbers of red cells (erythrocytes) and the amount of haemoglobin in the blood. There is, therefore, a greater capacity for the oxidation of lactic acid and fatty acids to CO_2 .

Nevertheless, training is associated with a decrease in insulin secretion, possibly a higher glucocorticoid secretion, larger amounts of muscle glycogen and blood glucose and, because of the greater work capacity, higher concentrations of blood lactate. The glucocorticoids, and possibly epinephrine in the trained animal, stimulate a more efficient breakdown (lipolysis) and oxidation of body fat as a source of energy, so conserving glycogen and yielding higher concentrations of NEFA in the blood. The glycerol released during fat breakdown tends to accumulate during hard exercise, possibly because of the raised concentration of blood lactate, and only on completion of hard muscular work is it utilized for the regeneration of glucose (Fig. 2.1).

The energy requirements of extended work can be accommodated entirely within the aerobic breakdown of glucose and by the oxidation of body fat. Thus, no continued accumulation of lactate was observed in two horses subjected to an endurance ride (Fig. 2.4), and although body fat represents the primary source of energy, its relatively slower breakdown means that there is a gradual exhaustion of muscle and liver glycogen associated with a continuous decline in blood glucose (Fig. 2.5), despite elevated concentrations of NEFA in the blood. Exhaustion occurs when blood glucose reaches a lower tolerable limit.

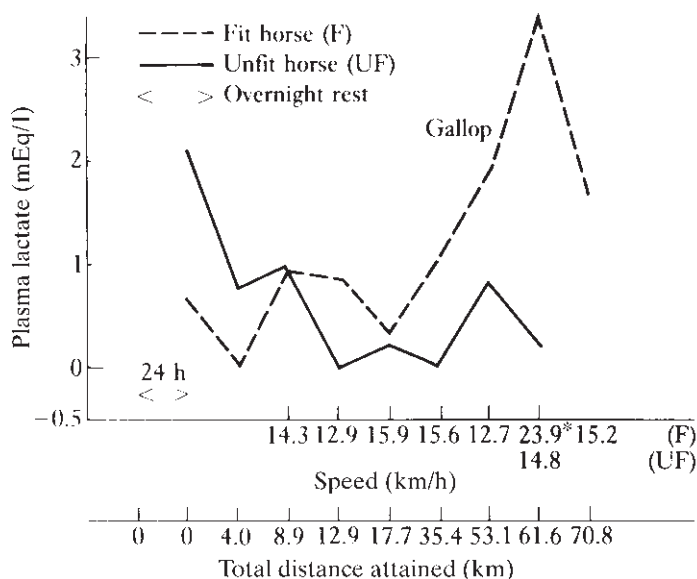


Fig. 2.4 Effect of speed but lack of effect of distance achieved on the concentration of plasma lactate. Horses separated at 53.1 km*. Note: only fit horse galloped between 53.1 and 61.6 km from start. Unfit horse retired after 61.6 km (Frape *et al.* 1979).

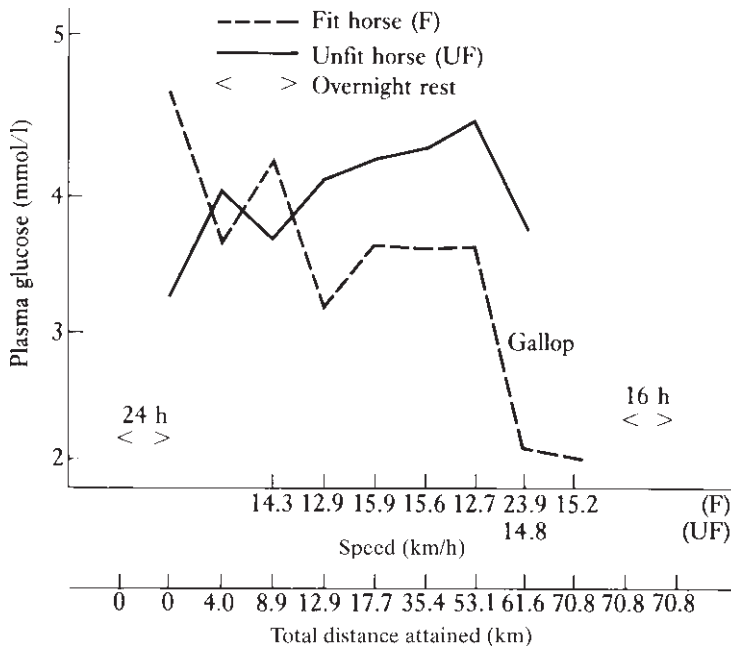


Fig. 2.5 Effect of speed and distance achieved on the concentration of plasma glucose in two horses (Frape *et al.* 1979).

In a more general sense, hypoglycaemia (low blood glucose) contributes to a decrease in exercise tolerance. Therefore, horses and ponies conditioned to gluconeogenesis – that is, the production of glucose from noncarbohydrate sources through adaptation and training (Fig. 2.1) – may more readily withstand extended work. Hypoglycaemia may occur when extra-hard exercise coincides with a peak in insulin secretion, suggesting that horses and ponies conditioned to gluconeogenesis through high-roughage diets, may more readily cope with sustained anorexia (persistent lack of appetite, usually through feed scarcity).

Glucose represents a much larger energy substrate in individuals given a high-grain diet, whereas VFAs will do so in those subsisting on roughage. Horses and ponies accustomed to a diet rich in cereals will have, in a rhythmical fashion, greater peaks and lower troughs of blood glucose than those individuals maintained on a roughage diet, owing to differences in insulin secretion and the differences in rates of consumption of the two types of diet. The grain-fed horse at peak blood glucose is more spirited, and less so in the trough, but cannot necessarily sustain work better at the peak. The practical corollary of this is that individual horses and ponies accustomed to a diet rich in concentrates should be fed regularly and frequently in relatively small quantities, not only to prevent the occurrence of colic, but also to smooth out the cyclic changes in blood glucose (preparation for exercise is a different matter and is discussed in Chapter 9). In Fig. 2.6 the energy transfers of the young adult working horse are summarized.

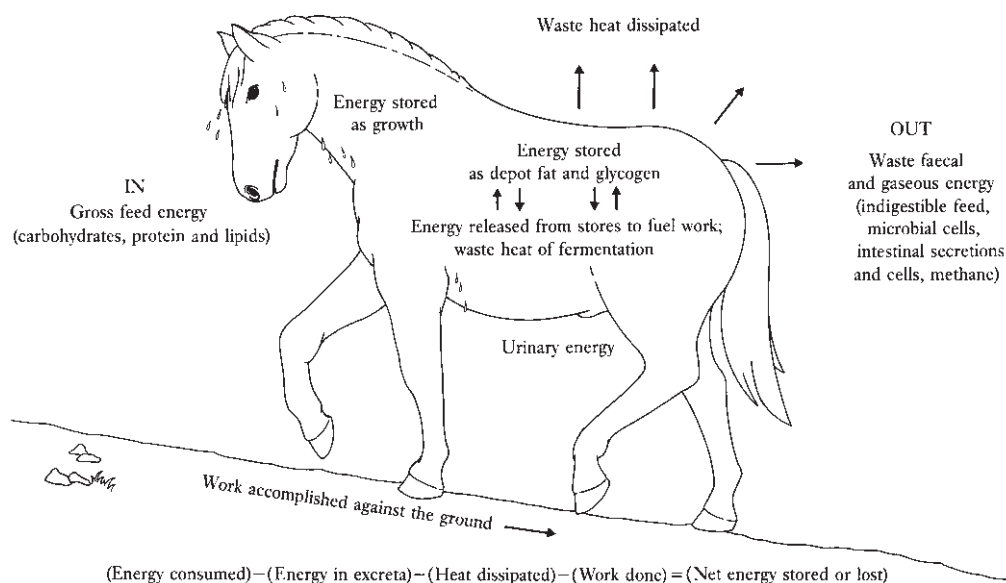


Fig. 2.6 Energy transfers in the young adult working horse.

Appetite

There is conflicting evidence about the factors that control appetite and hunger in horses and ponies. It is clear that amounts of NEFA in the blood are not significantly different between satiated individual animals and those with a normal hunger. It also seems that satiety is not directly associated with an elevation in blood glucose, although individuals with low concentrations of blood glucose tend to eat more and faster. Blood-glucose concentration in ponies may not influence the amount of food consumed in a meal, but it may influence the interval between voluntary feeds, without affecting the amount consumed when the pony goes to the feed trough. Supplementary corn oil seems to extend the interval before the next meal and reduce total feed intake 3–18 hours after administration.

A trigger mechanism controlling the feeling of satiety, or hunger, in the horse or pony may be the amount of digestion products (especially glucose) in the intestine and VFA production in the caecum. That is, when these products in the intestinal lumen and mucosa attain certain concentrations, eating stops, and this may be mediated by afferent vagal nerve fibres. With access to feed, eating recommences when these concentrations have fallen below a certain threshold. The degree of fill in the stomach and the blood-glucose concentration, according to this evidence, have no influence on eating; but taste, visual contact between horses, energy density of feed, rate of eating and time of day all seem to influence feed intake. The practical interpretation of this for feeding management is considerable and will be discussed in Chapter 6.

AMINO ACIDS

Proteins consist of long chains of amino acids, each link constituting one amino acid residue. In all the natural proteins that have been examined, the links, or α -amino acids, are of about 20 different kinds. Animals do not have the metabolic capacity to synthesize the amino group contained in half the different kinds of amino acid. The horse and other animals can produce certain of them from others by transferring the amino group from one to another carbon skeleton in a process known as transamination. Ten or eleven of the different types cannot be synthesized at all, or cannot be synthesized sufficiently fast, by the horse to meet its requirements for protein in tissue growth, milk secretion, maintenance etc. Plants and many microorganisms can synthesize all 25 of the amino acids. Thus, the horse and other animals must have plant material in their diet, or animal products originally derived from plant food, in order to meet all their needs for amino acids (i.e. they are unable to survive on an energy source and inorganic N). Whether or not microorganisms, chiefly in the horse's large intestine, synthesize proteins, the amino acids of which can be utilized directly by the horse in significant amounts, is still a contentious issue. The consensus is that although this source makes some contribution, probably in the small intestine, only small amounts of amino acid can be absorbed from the large intestine and by far the major part is voided as intact bacterial protein in the faeces.

During the digestion of dietary protein, the constituent amino acids are released and absorbed into the portal blood system. The amount of protein consumed by the horse may be in excess of immediate requirements and although there is some capacity for storing a little above those needs in the form of blood albumin, most excess amino acids, or those provided in excess of the energy available to utilize them in protein synthesis, are deaminated in the liver with the formation of urea. The concentration of urea rises in the horse's blood (Fig. 1.6), although some of the amino-nitrogen may be utilized in the liver for the synthesis of dispensable amino acids (Fig. 2.7). An increase in the blood concentration of urea in endurance horses may simply reflect rapid tissue protein catabolism for gluconeogenesis in glycogen depletion (Fig. 2.8). In addition, of course, the carbon skeleton of deaminated dietary glucogenic (e.g. glycine, alanine, glutamic acid, proline, methionine) and ketogenic (e.g. leucine and in part isoleucine, phenylalanine and tyrosine) amino acids is used as an energy source.

The extent to which dietary protein meets the present requirements of the horse depends on its quality as well as its quantity. The more closely the proportions of each of the different indispensable amino acids in the dietary protein conform with the proportions in the mixture required by the tissues, the higher is said to be the quality of the protein. If a protein, such as maize gluten, containing a low proportion of lysine is consumed and then digested, the amount of it which can be utilized in protein synthesis will be in proportion to its lysine content. As the lysine is limiting, little of it will be wasted, but, conversely, the other amino acids, both dispensable and indispensable, will be present in excessive quantities and so will be deaminated to an alarming extent.

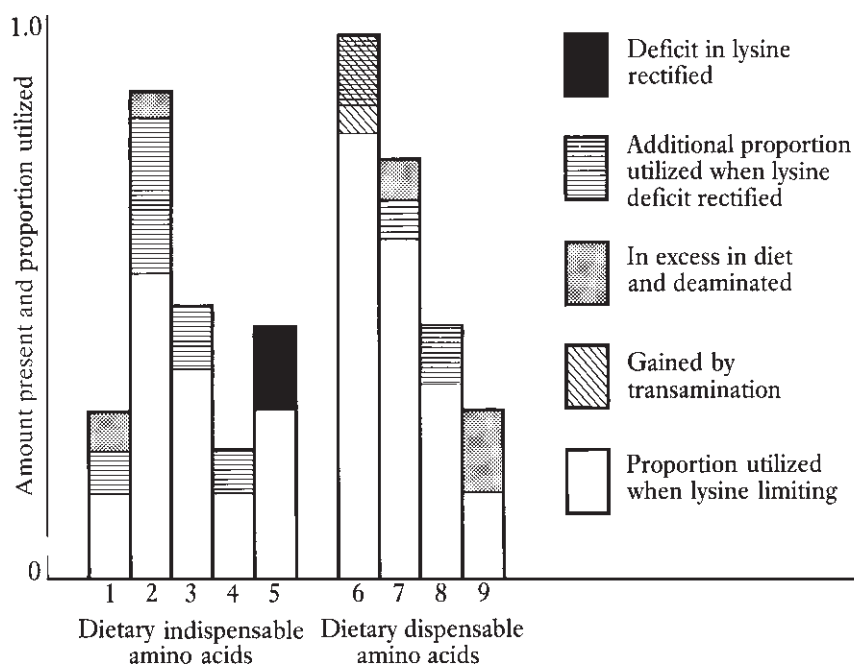


Fig. 2.7 Effect of lysine supplementation on the utilization of a dietary protein that is grossly limiting in its lysine (5) content. Only nine of a possible 25 amino acids are shown. Amino acids 3 and 4 limit further utilization of a lysine-supplemented diet. Addition of supplementary 3 and 4 now would decrease deamination of 1, 2, 7 and 9.

Synthetic supplements

If the relative deficiency of lysine in the gluten is made good by supplementing the diet with either a good-quality protein, such as fish-meal, or with synthetic lysine, then the amounts of each of the amino acids available in the blood plasma will more closely conform with the requirement, so that proportionately more of those amino acids can be used for protein synthesis (Fig. 2.7). It has been shown that the proportions of amino acids in the common sources of feed proteins given to horses and ponies are such that lysine is the indispensable amino acid most likely to limit the tissue utilization of the protein and threonine the second most likely.

Several studies have been undertaken with growing TB and Quarter Horses to measure their growth response to the addition of lysine-HCl to conventional 12% crude protein concentrates containing maize, oats and soya-bean meal, fed with hay. Graham *et al.* (1993) gave such a concentrate, to appetite, twice daily with coastal Bermuda grass hay (1 kg/100 kg BW) to yearlings for 112 days (Table 2.1). The concentrate was supplemented with 2 g/kg diet lysine or with 2 g/kg lysine plus 1 g/kg threonine, or neither. Amino acid supplementation increased the rate of body weight gain and the efficiency of gain, while decreasing serum urea content. This reduction would indicate that threonine improved the amino acid balance of the

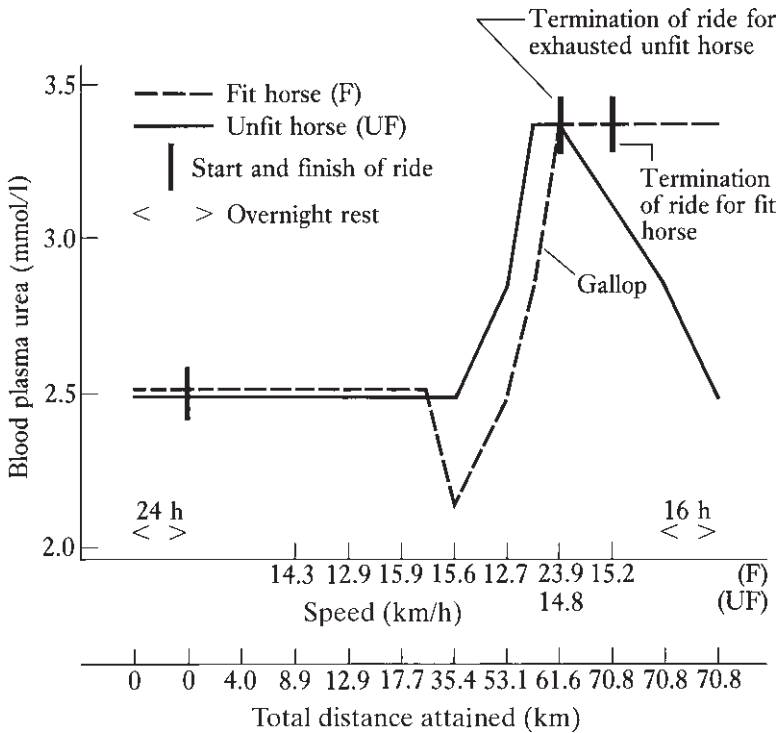


Fig. 2.8 Effect of speed and distance on the concentration of blood urea. The rise in plasma urea of the unfit horse resulted from a stress other than galloping (data from Frape *et al.* 1979).

Table 2.1 Treatment mean response (with SEs) of TB and Quarter Horse yearlings to amino acid supplementation of a concentrate mix (Graham *et al.* 1993).

	Gain per feed (g/kg DM)	Daily weight gain (g/day) (SE)	Girth gain (cm) (SE)	Urea N (mg/g serum (protein) (SE)
Basal	71.0	570 (20)	9.7 (0.49)	2.6 (0.20)
Basal + lysine	77.2	640 (20)	10.1 (0.46)	2.5 (0.19)
Basal + lysine + threonine	78.7	670 (20)	11.3 (0.47)	2.0 (0.19)

diet. Threonine increased the increment in muscle gain over the increment owing to lysine, as indicated by an absence of an increase in rump fat with the amino acid supplements.

Even lower protein pasture supplements have been used. Grazing TB mares and foals were given either a 14% CP mix composed of maize, soya-bean meal and corn oil, or a 9% CP mix supplemented with 6g/kg L-lysine and 4g/kg L-threonine (Staniar *et al.* 1999). Growth rates were similar over 12 months, although the latter group of foals grew faster in October, following weaning, when pasture quality was declining.

Inadequate dietary protein and hormone and neurotransmitter secretion

It has been proposed in several quarters that the NRC (1989) recommendations for protein in exercising horses are excessive, as the NRC consider that these requirements are proportional to energy requirements. Nevertheless, Wickens *et al.* (2003) observed that N retention was increased over a 14-day period by increasing the protein intake of exercised horses to 12.5% above the NRC (1989) recommendations. This may be an inadequate period and measure of assessment, as urinary N increased, and other influences on N metabolism during running, as described in Chapter 9, may be more relevant.

In support of Wickens *et al.* (2003), and using published data, Lawrence *et al.* (2003b) estimated the N retention (not accounting for losses in sweat) of exercising and sedentary horses to be 93 and 53 mg/kg BW, respectively. Moreover, supplementation of the diet of lightly exercised adult horses with 2.5 g lysine and 2.0 g threonine per kg diet increased their muscle mass over a 14-week period (Graham-Thiers *et al.* 2003).

Inadequate dietary protein causes a fall in the concentration of plasma albumin, total protein and, according to Scandinavian evidence, in the concentrations of plasma free essential amino acids: isoleucine, leucine, lysine, phenylalanine, threonine and valine (see Chapter 8). These changes severely restrict the rate of protein synthesis and may influence hormone secretion. Aspartic acid and glutamic acid are secretagogues for growth hormone and aspartic acid to some extent for gonadotropic hormones, whereas arginine and lysine are secretagogues for prolactin and insulin (Sticker *et al.* 1999). Tryptophan is a precursor of the neurotransmitter serotonin. Nevertheless, an oral dose of 50 g tryptophan in Standardbreds, two hours before exercise, was insufficient to induce a serotonin response, although maximum mean heart rate during exercise was decreased (Vervuert *et al.* 2003c).

Digestibility of protein

Another attribute of dietary protein that should not be neglected when alternative feeds are available is digestibility. For example, leather is a rich source of protein, but valueless to the horse because of its low digestibility. Most proteins have an apparent digestibility coefficient of 0.6–0.8, but this indicates only the amount of N digested. Protein that reaches the large intestine does not provide amino acids that are absorbed in significant amounts. Precaecal apparent digestibility of most amino acids is 0.3–0.6 (Almeida *et al.* 1999, and see also this book, Chapter 5), implying that small intestinal digestibility of amino acids is the critical criterion for comparing dietary sources. A feature related to that of digestibility is availability of amino acids and of lysine in particular. In practice, a reduction in lysine availability is encountered when skimmed milk, fish-meal and meat meals are overheated during processing. The excessive heating in milk leads to a reaction between some of the lysine and both unsaturated fats and the reducing sugar, lactose. These reactions lead to products from which the animal's digestive system cannot recover the lysine.

In summary, the protein value of a diet is the product of the amount, quality and digestibility of its constituent amino acids.

NON-PROTEIN NITROGEN

Urea is synthesized in the liver from amino acids present in excess of need so that a rise in dietary protein above requirements is associated with a rise in plasma urea (Figs 1.6, p. 27, and 2.7). In ponies given diets containing from 6% to 18% protein, between 200 and 574 mg urea N/kg metabolic BW ($W^{0.75}$) daily are recycled and degraded in the intestinal tract. In a pony weighing about 150 kg, this range is equivalent to 54–154 g crude protein daily.

While urea is within the tissues of a horse it cannot be degraded or otherwise utilized. However, when provided with an adequate source of dietary energy, microorganisms – chiefly in the large intestine – utilize it in protein synthesis, first degrading urea to ammonia by the action of bacterial urease. In the absence of an adequate supply of energy, which is normally present as fibre, starch and protein, a proportion of the ammonia, at a relatively neutral pH, diffuses back into the blood and may not be effectively utilized either by the horse or by its captive microorganisms (NH_3 -nitrogen may be incorporated by the liver into nonessential amino acids during transamination reactions, but this would not necessarily increase net N balance).

A fine balance is required, for in the absence of sufficient nitrogen, microbial growth cannot occur at a maximum rate and therefore a maximal rate of fibre breakdown and utilization will not prevail. Whereas circulating urea is nontoxic to the horse, except when very high concentrations affect osmolality, the absorbed ammonia is highly toxic. A healthy liver copes adequately with low concentrations in the amination of keto acids, forming dispensable amino acids, and by urea synthesis. However, if liver failure occurs, and this is more frequent in older horses, ammonia intoxication can occur without any increase in blood urea (see Chapter 11).

Limited evidence (Fig. 1.6, p. 27) does not support the widely held view that excessive protein consumption per se predisposes horses to laminitis. The flow of urea and other nitrogen compounds into the large intestine from the ileum varies with the quantity of diet and its type (Table 1.6). These digesta are relatively impoverished of nitrogen in horses receiving a diet of straw. The provision of non-protein nitrogen (NPN) or, for that matter, of protein as a supplement to this diet, results in an increased flow of nitrogen and a stimulation of microbial growth in the large intestine. Urea, or more effectively biuret, added to low-protein diets in concentrations of 1.5–3%, has increased nitrogen retention in both adult and growing horses with functioning large intestines, and pregnant mares subsisting on poor pasture apparently benefit from the consumption of supplementary urea.

Nevertheless, in most other circumstances the response to a urea supplement is poor and difficult to justify. Martin *et al.* (1996b) found that no nutritional benefit could be derived by mature horses from urea supplementation of a low-protein diet,

in that N balance was not increased. Where urea has been given to lactating mares the limiting factor has usually been energy intake. In this situation, feed intake and body weight have been depressed and plasma urea N has been increased without raising blood ammonia concentration.

The addition of urea or biuret to low-protein, poor-quality hay diets may increase DM and fibre digestion as well as N retention by stimulating microbial growth. These effects are, however, small from a practical viewpoint. Detailed studies with adult geldings, conducted by Martin *et al.* (1996b), failed to find any improvement in barley straw digestion, measured as dry matter, organic matter or neutral detergent fibre digestibility, from the addition of 20.3 g urea/kg dietary DM to a diet containing 4.4 g/kg N.

In summary, it would seem that horses and ponies with functioning large intestines and given diets containing less than 7–8% crude protein may make only minor use of supplementary NPN as an adjunct to that secreted back into the small intestine in digestive secretions and more directly from the blood. The reason for this is that bacterially synthesized amino acids are absorbed from the large intestine in only small amounts. In ruminants, large amounts of soluble N entering the rumen lead to a rapid production of ammonia and therefore to ammonia toxicity.

Treatment of ammonia toxicity

Ammonia toxicity, expressed as hyperammonaemia (blood levels exceeding 150 $\mu\text{mol/l}$; note that careful sample handling is required, with rapid analysis), caused by excessive dietary non-protein nitrogen, or protein, is less likely in the healthy horse with normal hepatic function, chiefly because much of the nitrogen is absorbed into the bloodstream before it reaches the regions of major microbial activity in the large intestine. Nevertheless, hyperammonaemia has been produced experimentally from the ingestion of large amounts of urea, but in these cases blood urea is also elevated. Where serum urea levels are normal (6–8 mmol/l), liver dysfunction is frequently the cause of hyperammonaemia with encephalopathic signs (ammonia readily crosses the blood–brain barrier to compete with K^+). Peek *et al.* (1997) reported evidence of hyperammonaemia associated with normal blood urea and liver enzyme levels, but with hyperglycaemia and acidaemia. Clinical signs included head pressing, symmetric ataxia, tachycardia and diarrhoea, and behaviour suggesting sudden blindness and abdominal pain.

Ammonia interferes with the citric acid cycle, oxidative phosphorylation and aerobic metabolism, resulting in lactic acidosis and hyperglycaemia. Treatment should therefore include administration of fluids, excluding dextrose, but including strong ions to counteract acidosis, given slowly intravenously. Where liver dysfunction has been eliminated as a cause, the origin of the ammonia is likely to be the large intestine. In this case, oral acidifying agents, such as lactulose, should also be given. They decrease ammonia absorption by converting it to the ammonium ion.

PROTEIN FOR MAINTENANCE AND GROWTH

Maintenance

Tissue proteins are broken down to amino acids and resynthesized during normal maintenance of adult or growing animals. This process is not fully efficient and, together with losses of protein in the sloughing of epithelial tissues and in various secretions, there is a continual need of dietary protein to make good the loss. However, these losses are relatively small in comparison with the protein synthesis of normal growth, or milk production, and proportionately less lysine is required. It follows that less protein, or protein of poorer quality, is needed for maintenance than is necessary for growth or milk secretion. Nevertheless, it has been shown that the protein needs of the adult horse for maintenance are less when good-quality protein is provided than when poor-quality protein is given. For example, adult TB mares were shown to remain in nitrogen balance when given 97 g fish protein/day, but they required 112 g for balance when the protein source was maize gluten.

The protein needed by the horse for body maintenance can be defined as the amount of protein required by an individual making no net gain or loss in body nitrogen and excluding any protein that may be secreted in milk. In these circumstances the animal must replace shed epithelial cells and hair, it must provide for various secretions and keep all cellular tissues in a state of dynamic equilibrium. The losses are a function of the lean mass of body tissues, depicted as a direct proportion of metabolic body size. For most purposes, the latter is considered to be the body weight (BW) raised to the power 0.75, and evidence suggests that horses daily require about 2.7 g digestible dietary protein/kg BW^{0.75}. A horse weighing 400 kg would therefore need daily about 240 g digestible protein, or 370 g dietary crude protein. This assumes that the protein has a reasonable balance of amino acids, although, as already pointed out, the lysine content of the protein for maintenance need not be as high as that required for growth (discussed in more detail in Chapter 6).

Growth

A young horse with a mature weight of 450 kg normally gains 100 kg between three and six months of age at the rate of 1 kg daily. Growth rate in kilograms per day declines during the succeeding months and it therefore gains the next 100 kg between about 6 and 12 months of age and 75 kg between 12 and 18 months (Hintz 1980a). From a very young age the rate of gain per unit of body weight decreases continuously, while the daily maintenance requirement increases (Chapter 8). As the weaned foal grows, an increasing proportion of that daily gain is composed of fat and a decreasing proportion is lean. It is thus apparent that the dietary requirement for protein and the limiting amino acid lysine decline with increasing age in the growing horse. For colts aged three months, a maximum rate of gain has been achieved with diets containing 140–150 g protein/kg and 7.5 g lysine/kg.

Diets may differ in the amount of digestible energy they provide per kilogram. For obvious reasons it is more accurate to state the protein requirements as a proportion of the digestible energy (DE) or net energy (NE) provided. Current evidence suggests that TB and Quarter Horse yearlings require 0.45 g lysine per MJ DE (Chapters 6 and 8). A compound stud nut for young growing horses may contain about 12–13 MJ DE/kg and oats about 11 MJ DE/kg. However, hard hay, containing 50–60 g protein/kg may provide 7.5–8 MJ DE/kg. If the yearling consumes a mixture (approximately 50 : 50) of concentrates and hay, the diet provides on average 10 MJ DE/kg and the minimum lysine requirement is 4.5 g/kg total diet (i.e. 0.45 g/MJ DE). Hay of 50–60 g/kg protein may contain only 2 g/kg digestible lysine and therefore the concentrate should contain at least 7 g lysine/kg in order to meet the minimum requirement. A yearling consuming 9 kg daily of total feed of this type would receive about 40 g lysine.

Much of the growth of horses may take place on pasture. Leafy grass protein of several species has been shown to contain 55–59 g lysine/kg. During the growing season the protein content in the dry matter of grass varies considerably from 110 to 260 g/kg in the leaf, whereas the flowering stem contains only 35–45 g/kg. Thus, the lysine content of the grazed material as a fraction of air-dry weight can vary from 5 to about 13 g/kg, and, if a leafy grass diet is supplemented with a concentrate mixture, the lysine and protein requirements may be met by cereals as a source of that protein. Because the quality of pasture varies so much, the use of cereals alone may mean that the protein and lysine requirements are not always met and, of course, the mixture may be inadequate as far as several other nutrients are concerned. Table 10.4, p. 375, gives some analytical data for pasture in several months of the grazing season.

Laminitis and energy intake

Increased risk of laminitis and colic have been associated with an abrupt overload of non-structural carbohydrate (NSC), attributed to hydrolysable carbohydrate (CHO-H) in grain concentrates as well as to rapidly fermentable carbohydrates (CHO-F_R) in pastures. Hoffman *et al.* (2001) demonstrated that CHO-H accounted for 97% or more of the NSC in concentrates, but for only 33% of it in pasture and hay, the remainder of NSC being CHO-F_R. These workers found that pasture was surprisingly rich in CHO-F_R during the autumn and this fraction of NSC is a major contributor to laminitis risk. Horses should be adjusted gradually to lush pasture by slowly increasing their daily period of access to allow the microbial population of the large intestine to adapt. Similar advice applies to concentrate feeding.

De Fombelle *et al.* (1999a) abruptly introduced 30% or 50% of the DM as barley into a hay diet, as part of two daily meals for ponies. At each meal the barley was consumed before the hay. The amount of starch did not exceed 2.3 g/kg BW per meal, but the colonic lactate concentration increased tenfold and the *Lactobacillus* and *Streptococcus* populations were increased in the right ventral colon five hours following the diet change, without a significant decrease in the pH. Fourteen days

after adaptation to the 50% barley the large intestinal pH had dropped from 6.74 to 6.26, associated with a decline in the population of cellulolytic bacteria. A consequential reduction in the digestion of fibre could create a favourable situation for large intestinal impaction (Reeves *et al.* 1996) (a method for *ad libitum* feeding of concentrates is outlined in Chapter 8 and laminitis is discussed in detail in Chapter 11). As horses utilise dietary fat efficiently, the gradual introduction of a diet rich in fat and fibre should play a useful role in prevention of digestive and metabolic disorders (Williams *et al.* 2001b).

Laminitis control

The laminitic risks associated with body-fat accumulation in overweight animals should be appreciated. Feed intake should be reduced gradually to decrease fat deposition, consistent with the avoidance of hyperlipidaemia. For horses and ponies of both normal- and high-condition score the selection of feeds suitable for laminitis requires analytical evidence.

General requirements of analytical method

The analytical method should be capable of estimating the following in feed:

- total starch* plus indigestible, rapidly fermentable carbohydrate (CHO-F_R); and
- neutral detergent fibre (NDF). (Improved analytical methods for fibre have been discussed and, no doubt, will be gradually introduced, see McCleary 2003, and associated papers).

The procedure of Hoffman *et al.* (2001) (Fig. 2.9) will allow these characteristics of the feed to be estimated from determinations of the moisture, crude protein, fat, ash and NDF. The non-structural carbohydrate (NSC) may then be calculated by summing these five determinants and subtracting from 100. Average NDF values of feeds are given by NRC (1989) and some will be found in Appendix C.

The NSC consists of hydrolysable carbohydrates (CHO-H) and rapidly-fermentable carbohydrate (CHO-F_R). The majority of CHO-F_R would reach the large intestine, whereas the proportion of CHO-H reaching the large gut would depend on:

- its digestibility; and
- the quantity of feed given in a meal.

If the feed material contains a small quantity of mono- and disaccharides (excepting lactose), which are normally digested in the small intestine, they may be extracted with boiling water. The remaining CHO-H may then, if necessary, be determined following enzymatic hydrolysis of starch. The value of (CHO-F_R + CHO-H) should

* It is informative to have an estimate of the fraction of starch that is digestible, the remainder being 'resistant' starch (resistant to hydrolysis by digestive enzymes).

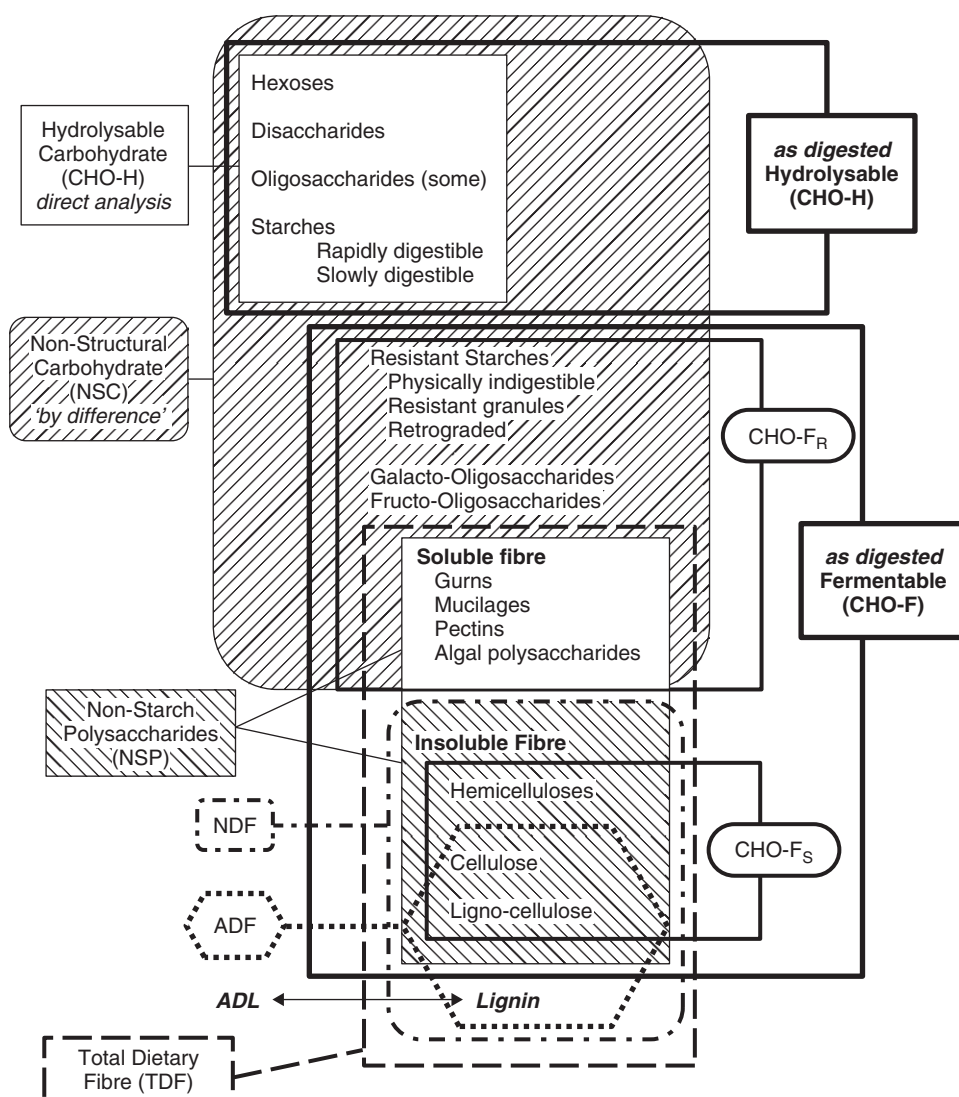


Fig. 2.9 A scheme of dietary carbohydrate fractions for the horse taken from Hoffman *et al.* (2001). Proximate analysis fractions of the diet are noted on the left of the figure. NSC is estimated as a difference: $NSC = 100 - (\text{water} + \text{protein} + \text{fat} + \text{ash} + \text{NDF})$. The fraction, Total Dietary Fibre, is frequently mentioned in descriptions of foods for human consumption. The fractions, as used by the horse, digestible carbohydrate (CHO-H) and fermentable carbohydrate (CHO-F), are noted on the right of the figure. CHO-H is the carbohydrate fraction that is digested by the horse with the release mainly of glucose, unless excessive amounts of starch are given in a meal, whereas resistant starches will pass to the hind-gut and together with excess CHO-H will constitute a risk of digestive upset. CHO-F is further subdivided into a rapidly fermented fraction (CHO-F_R), contributing to the risk, and a slowly fermented fraction (CHO-F_S). Reproduced with permission, Hoffman *et al.* 2001.

not exceed 0.25% body weight per meal to provide a reasonably low risk of diet-related laminitis.

It is recognised that pasture, as a feed source, presents major issues, in both definition of its chemical characteristics and quantity grazed per hour. In order to prevent excessive grazing it is necessary for the horse to eat an adequate amount of a safe feed before it is released onto pasture. This can be difficult to achieve. Evidence indicates that fructans (and possibly other indigestible oligosaccharides, all of which are components of CHO-F_R) present in pasture plants are a major cause of laminitis. It has been estimated that a 500 kg horse at pasture would on average consume 1.8 kg of readily fermentable CHO-F_R per day.

In respect of laminitis risk, safe feeds include:

- good quality hay;
- nutritionally improved straw (NIS);
- cereal straw;
- oat feed;
- soya hulls; and
- vegetable oil.

Feeds acceptable in restricted quantities:

- dried lucerne and grass pellets;
- molasses;
- wheat bran; and
- sugar beet pulp.

STUDY QUESTIONS

- (1) The horse evolved as a browsing animal engaging in many small feeds each day. What impact has this had in respect of:
 - (a) food selection and metabolic responses;
 - (b) social habits; and
 - (c) safety in the wild?
- (2) What is meant by limiting dietary amino acid and what is the relation, if any, between this and (a) maintenance diets and (b) production diets? What is meant by nitrogen balance?
- (3) What factors should be considered when a horse has lost its appetite for sufficient feed to maintain body weight?

FURTHER READING

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Chapter 3

The Roles of Major Minerals and Trace Elements

Grass is the first nourishment of all colts after they are weaned. . . . Whereas when they are fed with corn and hay, but especially with the first, . . . it exposes them to unspeakable injuries.

W. Gibson 1726

MAJOR MINERALS

Calcium (Ca) and phosphorus (P)

Function

The functions of calcium and phosphorus are considered together because of their interdependent role as the main elements of the crystal apatite, which provides the strength and rigidity of the skeleton. Bone has a Ca:P ratio of 2:1, whereas in the whole body of the horse the ratio is approximately 1.7:1.0, because of the P distribution in soft tissue. Bone acts as a reservoir of both elements, which may be tapped when diet does not meet requirements. The elements of bone are in a continual state of flux with Ca and P being removed and redeposited by a process that facilitates the reservoir role and enables growth and remodelling of the skeleton to proceed during growth and development. The acute role of Ca relates to its involvement in a soluble ionic form for nerve and muscle function. Consequently $[Ca^{2+}]$ concentration in the blood plasma must be maintained within closely defined limits.

Control of plasma Ca ion concentration and Ca and P metabolism

The flux and distribution of Ca and P in the body are strictly regulated by two proteinaceous hormones in particular, functioning antagonistically at the blood–bone interface, the intestinal mucosa and the renal tubules (see also under ‘Vitamin D’, Chapter 4). The two hormones are the potent parathyroid hormone, secreted by the parathyroid glands adjacent to the thyroid glands, and the less significant calcitonin, secreted by the parafollicular cells of the thyroid gland. A slight decrease in $[Ca^{2+}]$ concentration in extracellular fluid of horses causes the immediate secretion of parathyroid hormone (Estepa *et al.* 1998) and the stress of pregnancy and lactation cause the enlargement of the parathyroids. Excessive $[Ca^{2+}]$ concentration, as occurs in vitamin D toxicity, leads to a decreased activity and size of the glands. Calcitonin, on the other hand, rapidly decreases blood Ca ion concentration by decreasing osteoclastic, and increasing osteoblastic, activities.

Table 3.1 Mean values and ranges for serum total concentrations (mmol/l) of electrolytes in horses of different ages. (Modified from published tables of S.W. Ricketts, Beaufort Cottage Laboratories, Newmarket, Suffolk.)

		Birth to 36 h	3 Weeks	Yearlings	Horses in training	Mares at stud
Ca	Mean	3.2	3.2	3.3	3.4	3.4
	Range	2.7–3.6	2.5–4.0	2.7–4	2.6–3.9	2.9–3.9
P	Mean	2.5	2.5	1.8	1.3	1.1
	Range	1.2–3.8	1.6–3.4	1.4–2.3	1.1–1.5	0.5–1.6
Na	Mean	136	137.5	138.5	138.5	138.5
	Range	126–146	130–144	134–143	134–143	134–143
K	Mean	4.8	4.5	4.3	4.3	4.3
	Range	3.7–5.4	3.6–5.4	3.3–5.3	3.3–5.3	3.3–5.3
Mg	Mean	0.83	0.81	0.78	0.78	0.78
	Range	0.57–1.10	0.66–1.10	0.62–1.10	0.62–1.10	0.62–1.10
Cl	Mean	—	—	—	—	—
	Range	Normal range for all ages 99–109				

The horse kidney seems to play a greater part in controlling concentrations of Ca in the blood than does the intestinal tract and this may have practical significance for diet and renal disease. The mean values and ranges for serum total Ca and P, among others, are listed in Table 3.1. It should be noted that normal resting plasma phosphate concentration decreases with increasing age and that ionized plasma Ca concentration is approximately 1.7 mmol/l lower than the total values given in the table.

Serum phosphate values vary without untoward physiological effects. For example, strenuous exercise can depress blood phosphate to half the resting value for 2–2.5 hours. Nutritional secondary hyperparathyroidism (NSHP) is a diet-related clinical disorder of horses in which serum phosphate is slightly raised and serum $[Ca^{2+}]$ values are slightly depressed (Figs 3.1 and 3.2).

NSHP is well recognised, but is much less common today. Its typical presentation is fibrous osteodystrophia of the facial bones ('big-head'), present in a pony I once purchased from the Welsh hills. Nevertheless, NSHP occasionally occurs without the typical facial abnormality (Little *et al.* 2000). Other clinical signs include a stiff gait, or shifting-limb lameness, abnormal mastication with oral dysphagia and upper airway stridor.

Ca and P in bone

Horses have no 'horse sense' when it comes to selecting a diet containing a balanced mixture of Ca and P – they prefer the palatability of a P-rich diet, whereas this is not available for selection in the natural grazing environment. Hence, a dietary Ca deficiency is not an infrequent occurrence among domestic horses.

Inadequate dietary Ca and P for growing foals causes a delay in the closure times of the epiphyseal plates of long bones and contributes to developmental orthopaedic

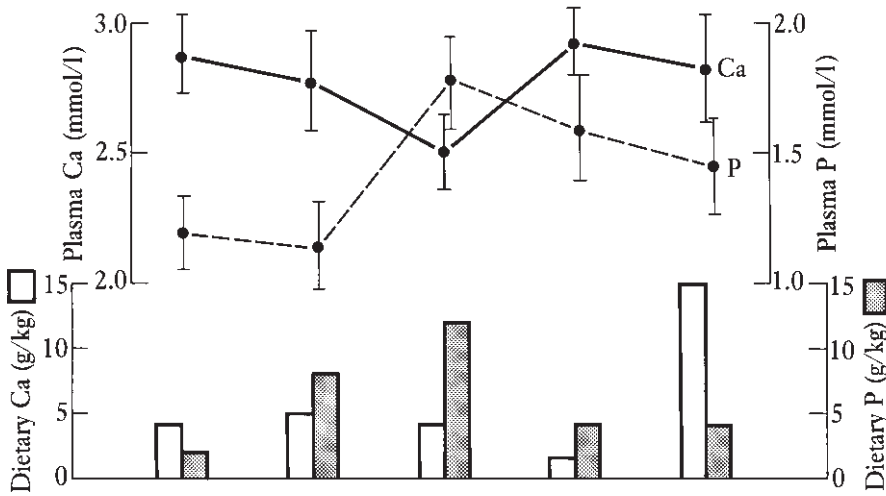


Fig. 3.1 Effects of dietary Ca (□) and P (■) on mean concentrations in blood plasma, $\pm$ SE (after Hintz & Schryver 1972).

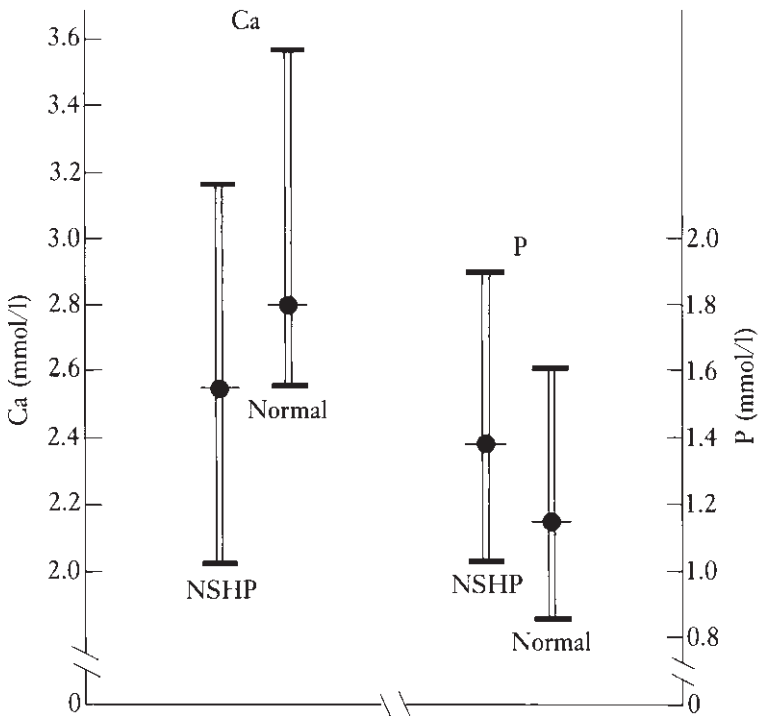


Fig. 3.2 Mean and range of serum Ca and P concentrations in spontaneous NSHP (Krook 1968). Notice how in cases of NSHP Ca is depressed and P elevated.

dic disease (DOD). In adult working horses it causes lameness and bone fractures. Failure of the osteoid, or young bone, to mineralize is called rickets in young and osteomalacia in adult horses (some authorities argue that true rickets does not occur in the foal).

In extreme cases, when mineral is being reabsorbed from bone, the outcome may be generalized osteodystrophia fibrosa in which fibrous tissue is substituted for hard bone as occurs in the facial bones ('big-head'). In the presence of vitamin D in each of these conditions the body, through the agency of parathyroid hormone (PTH), is endeavouring to maintain homeostasis of blood Ca by accelerating the removal of Ca from the bones and increasing the tubular reabsorption of Ca. Diets based on wheat bran and cereals are rich in organic P and low in Ca, predisposing horses to these conditions.

The tendency to lower blood $[Ca^{2+}]$ leads to increased bone resorption, increased renal excretion of phosphate, an increased rate of bone–mineral exchange and to a greater susceptibility of bones to fracture. Deposition of Ca salts in soft tissue, including the kidney (nephrocalcinosis), may also be apparent. A physiological example of rising serum PTH concentration occurs in periparturient mares during mammary secretion of Ca when there is a decrease in serum total and ionized Ca concentrations (Martin *et al.* 1996a), and an active fragment of PTH has been detected and measured in mare's milk (Care *et al.* 1997). The hormone calcitonin, or thyrocalcitonin as it is secreted by the parafollicular C cells of the thyroid gland, opposes the effects of PTH. When plasma Ca concentration is elevated, PTH secretion is decreased, reducing plasma Ca by decreasing the activity of osteoclasts and increasing that of osteoblasts.

Hypocalcaemia

(See also Chapter 9.) Ca is principally an extracellular cation existing in ionized $[Ca^{2+}]$, complexed and protein-bound forms. The ionized form is physiologically active and its concentration in blood plasma is precisely controlled, but the concentration is influenced by acid–base changes. Normal serum total Ca concentration in the horse is 3.2 mmol/l and ionized Ca concentration is about 1.5 mmol/l, although laboratory method influences the values obtained.

Pronounced hypocalcaemia is unlikely to result from a dietary inadequacy of Ca, but rather from metabolic alkalosis. Thus, hypocalcaemia may occur in adult horses as a post-exertional stress. The clinical signs are: muscle fasciculation and tetanic spasms, incoordination, synchronous diaphragmatic flutter, decreased gut sounds and even inability to stand. Extended work and overheating lead to a rise in blood pH that depresses the concentration of $[Ca^{2+}]$ in the blood. Moreover, elevated body temperature per se can bring about a loss of 350–500 mg Ca/hour in sweat and this rate of loss may exceed the capacity of blood replenishment by bone mobilization.

Fast exercise causes a fall in blood pH and is generally expected to be associated with a slight rise in plasma $[Ca^{2+}]$; but, surprisingly, Vervuert *et al.* (2002, 2003b)

detected a fall in plasma pH and $[Ca^{2+}]$, but rises in lactate, P_i (inorganic P) and intact PTH. Low-speed exercise resulted in an increase in pH, whereas lactate, $[Ca^{2+}]$, total Ca, P_i and PTH were unchanged. Nevertheless, in accord with the assumed mechanism for homeostatic control of plasma ionised Ca, a close negative relationship between intact PTH and $[Ca^{2+}]$ was maintained (Vervuert *et al.* 2002). Intact PTH is a mediator in counter-regulation of exercise-induced hypocalcaemia by reabsorption of renal Ca or by osteoclast-mediated bone resorption.

Van der Kolk *et al.* (2002) determined the normal range of $[Ca^{2+}]$ values for healthy horses to be 1.45–1.75 mmol/l heparinized blood and 1.58–1.90 mmol/l serum. (Spanish work showed that when plasma $[Ca^{2+}]$ fell to 1.25 mmol/l, PTH attained a maximum of >80 pg/ml, but, when plasma $[Ca^{2+}]$ rose to 1.8 mmol/l, PTH dropped to a minimum of <12 pg/ml). Van der Kolk *et al.* (2002) showed that the average EDTA plasma concentrations for intact PTH and for C-terminal PTH were respectively 0.6 pmol/l and 96 pmol/l. Spanish work, on the other hand, indicated a plasma PTH concentration of 20–30 pg/ml, determined by radioimmune assay, employing an antibody against intact human PTH, and 35–45 pg/ml with an antibody against the amino-terminal region of rat PTH. This fragmented region possesses biological activity. Van der Kolk's values for intact PTH are lower than the Spanish values (0.6 pmol/l $\equiv$ 5.7 pg/ml, human PTH, MW 9424).

As $[Ca^{2+}]$ concentration varies with blood pH, Van der Kolk computed a prediction equation for heparinized blood at pH 7.4:

$$[Ca^{2+}]_{pH7.4} \text{ mmol/l} = -6.4570 + 0.8739 (\text{pH}_{\text{measured}}) + 0.9944 ([Ca^{2+}]_{\text{measured}} \text{ mmol/l})$$

The intravenous (i.v.) administration of a solution of 50–100 mmol $CaCl_2$, or Ca gluconate, in 1 l over 30 min should resolve the clinical signs of hypocalcaemia. The higher dose may cause cardiac arrhythmias and so administration should be accompanied by cardiac auscultation, decreasing the rate of dosing if necessary. Hypercalcaemia also induces cardiac arrhythmias (hypo- and hypercalcaemias, respectively, prolong and shorten the Q–T interval of the electrocardiogram).

Dietary levels of Ca and P in relation to requirement

Excessive amounts of dietary Ca do not seem to initiate the kinds of dietary problems encountered in other domestic species. However, one experiment in which Shetland foals aged four months were given a diet containing 25 g Ca/kg, with a Ca:P ratio of 6:1, for four years (Jordan *et al.* 1975) resulted in a slight enlargement of the marrow region of the long bones and thinning of the cortical area, together with less bone mineral per unit of cortex. Lameness through loss of support for tendons and ligaments is frequently a characteristic of resorption of cortical bone. Excessive amounts of dietary Ca may make bones brittle, through abnormal bone storage of Ca. However, investigations in which diets containing 7–27 g Ca/kg have been compared for up to two years show that differences are small; bone density is increased by high-Ca diets and the cortex of the long bones is slightly thinner (Schryver *et al.* 1970a, 1971b, 1974a, 1974b). Grace

Table 3.2 The response of yearling Quarter Horses between 12 and 18 months of age to diets containing 115%, 100% and 85% of the NRC (1989) dietary requirements for calcium (Moffett *et al.* 2001).

	12 months	15 months	18 months
115%			
Intake, Ca/day g	28.13	32.87	38.00
Apparent digestibility, %	39.75	42.98	40.36
Retention, g/day	8.07	12.86	11.64
100%			
Intake Ca/day, g	25.35	29.14	39.40
Apparent digestibility %	38.75	38.89	55.85
Retention, g/day	6.90	9.93	18.47
85%			
Intake Ca/day, g	19.33	21.05	36.60
Apparent digestibility %	38.61	39.97	64.03
Retention, g/day	2.74	7.49	18.87

et al. (1998b) measured the mineral composition of horses aged 150 days (approximately 190 kg BW) and assuming a growth rate of 1.0 kg/day on pasture estimated the daily requirement for Ca and P to be 28 g and 21 g/day respectively.

Rapid growth rate has questionable consequences. Weanling stock-type horses 187 days of age and 236 kg BW were fed to increase at 0.97 kg/day or 0.49 kg/day (Petersen *et al.* 2001) on diets that met, or exceeded, NRC (1989) mineral requirements. Growth at the faster rate caused a greater increase in bone mineral content, without any compromise of bone quality. Nevertheless, three of six of these horses developed clinical signs of physitis, accompanied by joint pain, demonstrated by knuckling over at the knee, from which they recovered by 302 days of age.

It has been generally shown that bone strength increases with increasing density and Ca content. Some precision has, therefore, been given to requirements by measurements of Ca retention. The increase of Ca intake to 115% of NRC (1989) recommendations (4.8 g Ca/kg diet, 11.7 MJ DE/kg) in growing horses increases Ca retention (Moffett *et al.* 2001) (Table 3.2).

Exercise is critical for bone strength and mineral content in growing horses (Henry *et al.* 2003). It has been shown to increase size and mass of the third metacarpal and serum carboxy-terminal propeptide of type I procollagen (PICP) concentration of weanling Quarter Horses (Williams *et al.* 2003a). For these reasons it is probable that pasturing, as opposed to stalling (Bell *et al.* 1999), or holding in dry lots (Stephens *et al.* 2003) increases bone mineral content of young horses.

The bone density of juvenile Quarter Horses in training was increased by giving them moderately large amounts of Ca, P and Mg (respectively 151%, 130% and 159% of NRC 1989 recommendations) cf. lower amounts (respectively 136%, 98%, and 126% of NRC 1989) (Nolan *et al.* 2001). Nevertheless, despite higher Ca intakes than in those described by Moffett *et al.* (2001, Table 3.2), during the early stages of training, in long yearlings and two year olds, there is apparently a period of demineralization of bone with a decrease in Ca and P retention, but not in that of Mg

(Stephens *et al.* 2001), so that the stress of hard exercise should be delayed. This demineralization is followed by a period of remineralization.

Bone turnover, including osteoblastic activity, may be tracked with serum osteocalcin. Bone resorption, i.e. osteoclastic activity, may be tracked by measuring the carboxy-terminal telopeptide of type I collagen (ICTP) and bone formation is monitored by measuring the carboxy-terminal propeptide of type I procollagen. It should be noted, however, that circadian changes in the serum concentrations of osteocalcin and IGF-I (which regulates bone turnover) of two-year-old TB mares have been shown. The peak time for osteocalcin was 0900 hours, and that for IGF-I, with less amplitude, was 1730 hours (Jackson *et al.* 2003). Amongst horses given 275% of NRC (1989) requirements for Ca and P, cf. those given 133% of the requirements, the Ca concentration was higher amongst geldings of various ages (2 to >15 year olds) given the greater quantity during both sedentary and exercise periods (Buchholz *et al.* 1999) and blood ICTP concentration was increased, indicating that bone remodelling was accelerated (Mansell *et al.* 1999). Juvenile racehorses given Ca, P and Mg intakes that were, respectively, 169%, 132% and 168% of NRC (1989) recommendations achieved higher rates of bone formation, with lower rates of demineralization, than did those given lower amounts (Michael *et al.* 2001). The revised mineral requirements are given in Table 3.3.

The equine kidney plays a vital part in Ca homeostasis, and daily urinary Ca excretion shows a direct relationship with intake. In many species the urinary loss of Ca is raised by increased intakes of sodium, disrupting Ca homeostasis. However, this relationship does not hold in the horse (see Sodium, this chapter). Diets rich in Ca yield urine containing a precipitate of Ca salts; the urinary loss of Ca in a 300 kg yearling given a diet containing 20 g Ca/kg was 20–30 g in 6–8 l of urine daily (that is, 0.36% Ca). The absence of calculus formation in the kidney demonstrates the horse's ability to deal with large amounts of Ca despite the low solubility of the element; conversely, a dietary deficiency of Ca yields urine almost devoid of the element.

In contrast, endogenous loss of Ca in faeces, representing the minimal obligatory loss that must be replenished from dietary sources, is largely unaffected by the dietary amounts. Endogenous faecal Ca and P have been estimated, respectively, to be 36 mg/kg BW and 18 mg/kg BW daily in growing Quarter Horses (Cymbaluk *et al.* 1989b). Urinary losses of Ca decrease by 50–75% in extended work (Schryver *et al.* 1975, 1978a), whereas sweat losses increase. During 20 min of hard work, ranges of 80–145 mg Ca and 11–17 mg P have been found in the total yield of sweat (Schryver *et al.* 1978c). Over a full day's hard work this source represents a considerable loss of Ca. On the other hand, horses and ponies idle for long periods retain less Ca than those worked, when the dietary P concentration is excessive. Following such an inactive period dietary Ca and P should be raised 20% above minimal requirement levels.

Horses must absorb about 2.5 g Ca/100 kg BW daily to balance the obligatory loss. Absorption rate of good-quality limestone is approximately 50% indicating required daily intakes of 5 g/100 kg BW. However, the accumulating evidence given

above indicates that much higher intakes increase bone density and strength in growing and young adult horses (Table 3.3).

Intestinal absorption

The lack of impact of dietary Ca on the efficiency of P absorption in the horse may be related to the fact that Ca and P are absorbed from different regions of the intestine (Fig. 1.5, p. 25). However, vastly excessive intakes of Ca increase the faecal loss of P and dietary Ca can affect absorption of other elements. For example, excessive Ca can depress the absorption of magnesium, manganese and iron owing to competition at common absorption sites, or possibly to the formation of insoluble salts. Meyer and colleagues in Germany have reported that 50–80% of dietary Ca and 45–60% of magnesium are absorbed in the small intestine (Meyer *et al.* 1982c), whereas there is a net secretion of these elements into the large intestine.

True Ca and P digestibilities are reported to decline from 71% to 42% and from 52% to 6%, respectively, between 6 and 24 months of age (Cymbaluk *et al.* 1989b). Aged horses may be even less efficient in the absorption of Ca. Meyer's group further demonstrated that the site of P absorption varies with composition of diet. No P is absorbed in the upper small intestine of horses fed solely on roughage, whereas some is absorbed in the distal small intestine, especially in those given only concentrates. Large amounts of phosphate secreted into the caecum and ventral colon probably act as a buffer to VFAs produced there, and the dorsal colon and small colon are the major sites of absorption and reabsorption of phosphate (Fig. 1.5, p. 25).

Availability

The net availability of Ca in a variety of feeds has been estimated to lie between 45% and 70%, except where significant amounts of oxalates are present. The dietary level of phosphate influences Ca absorption. When dietary P, as inorganic phosphate, was raised from 2 to 12 g/kg, Ca absorption was decreased by more than 50% in young ponies receiving a diet otherwise adequate in Ca (4 g/kg diet) (Schryver *et al.* 1971a). Dicalcium phosphate-P or bone flour-P is digested to the extent of 45–50%. Rock phosphate and metaphosphates are poor sources of P and Ca. Phosphorus in salts of phytic acid, the predominant source in cereal and legume seeds, is only 35% available, despite the presence of large numbers of intestinal bacteria secreting phytase. Phytate-P constitutes at least 75% of the total P in wheat grain and 54–82% of the P in beans. Phytase supplementation of diets containing 1.8–3.0 g P/kg and based on maize, oats, soya and grass hay failed to increase P digestibility in mature horses (Morris-Stoker *et al.* 2001; Patterson *et al.* 2002).

Large amounts of vitamin D in the diet can increase the utilization of phytin-P, but as these amounts are almost toxic they cannot be recommended. Phytin-P use may be improved by the addition of yeast culture to the diet (see 'Probiotics', Chapter 5). Experiments in pigs have shown that the addition of phytase, derived from *Aspergillus niger*, to the diet increased phytate digestion. Generally, most dietary P is of plant origin, which has a lower availability than most sources of Ca (excluding those rich in oxalates). Therefore growing horses may sometimes receive

inadequate available P in their diet for normal growth. Pagan (1989) has also argued that the endogenous P losses by young horses are about double those (20 cf. 10 mg/kg BW) used by the National Research Council (NRC) in estimating the dietary P requirement. Thus, with some diets, relying on their natural P content, the P requirement of young horses may not be met.

Oxalates and other dietary factors affecting Ca absorption

The bioavailability, or true digestibility, of dietary Ca varies considerably. The principal factors controlling bioavailability are:

- amount of dietary Ca (true digestibility is 0.7 at requirement intake, cf. 0.46 at several times the requirement);
- amount of dietary P (10 g P added/kg diet containing 4 g Ca/kg reduced true Ca digestibility from 0.68 to 0.43);
- vitamin D status (of less significance for absorption in the horse than in some other domestic species);
- dietary phytates and oxalates (phytates and oxalates bind Ca and so reduce Ca availability; Ca:oxalate < 0.5:1 causes NSHP; implicated grasses rich in oxalates include: napier, guineagrass, buffel [*Cenchrus ciliaris*], pangola [*Digitaria decumbens*], green panic, paragrass, kikuyu [*Pennisetum clandestinum*], setaria [*Setaria sphacelata*] and probably some species of millet grass; lucerne (alfalfa) contains oxalic acid, see below); and
- age of animal (bioavailability may decline slightly with age, but the relationship is not pronounced in horses).

Oxalic acid binds divalent cations, such as Ca, in an unavailable form. Many tropical grasses are rich in oxalates and their feeding is associated with osteoporosis and lameness in horses. In contrast, rumen microorganisms degrade oxalic acid so that plasma Ca concentration is unaffected and renal function remains unimpaired in sheep and goats (Duncan *et al.* 1997). Ca deficiency ailments are noticeable in horses grazing pastures, or given hay, containing an abundance of these species of tropical and subtropical grass (see also Chapter 10). The bioavailability of Ca may, nevertheless, differ among plant sources containing oxalates. Lucerne contains these compounds, but its Ca has a high feeding value, and about three-quarters of the Ca is absorbed, although the Ca content relative to that of oxalic acid is much higher than that in the grasses listed above. Lucerne Ca is better utilized than that from timothy hay, which is not a significant source of oxalic acid. Evidence in laboratory animals shows that the feeding of Ca sources that have a low bioavailability compromises both the quantity and quality of bone, whereas inadequate quantities of highly available Ca reduce only the quantity of bone. Whether this is a contributory factor to equine lameness is unknown.

In summary

The maintenance requirement for the major minerals Ca and P is that necessary to balance losses in the faeces and urine, as well as unspecified 'dermal losses'. There

Table 3.3 Minimum daily requirements for Ca and P of growing horses (Schryver *et al.* 1974a; Grace *et al.* 1998b; Michael *et al.* 2001; Moffett *et al.* 2001; Nolan *et al.* 2001; Petersen *et al.* 2001).

Age (months)	BW (kg)	Weight gain (kg/day)	Ca (g/day)	P (g/day)
3	100	1	55	31
6	200	0.8	55	29
12	300	0.6	50	28
18	375	0.3	50	28
Mature	450	0	46	27

is an additional need for growth and, in the breeding mare, for mineralization of the foetal skeleton and for lactation. Each kilogram of lean body tissue in the horse contains about 20g Ca and 10g P; the amounts required in the diet to allow for maintenance and growth are shown in Table 3.3. Mare's milk contains on average about 900mg Ca and 350mg P/kg (Fig. 7.3, p. 254). A 500kg mare may produce a total of 2000kg milk in a lactation extending over five to six months – a total lactation deficit of 1.8kg Ca and 0.7kg P derived from skeletal reserves and feed. For milk synthesis, dietary Ca and P daily requirements, with average availabilities of 50% and 35%, respectively, are 10g Ca and 5.5g P to balance that secreted daily. Limestone flour and dicalcium phosphate are reliable sources of Ca and the second of P also. The requirement of 10g Ca is met by 28g limestone or 40g dicalcium phosphate, which also meets the P needs entirely.

Magnesium (Mg)

Magnesium is a vitally important ion in the blood; it forms an essential element of intercellular and intracellular fluids, it participates in muscular contraction and it is also a cofactor in several enzyme systems. Bone ash contains 8g Mg/kg in addition to 360g Ca/kg and 170g P/kg. There is a small net absorption of Mg from the large intestine, but the majority of net absorption occurs from the lower half of the small intestine. The 'obligatory loss' of Mg secreted into the intestinal tract amounts to about 1.8mg/kg BW daily; a further obligatory loss of about 2.8mg/kg BW occurs in the urine, and the maintenance requirement to offset these losses is about 13mg/kg BW daily, or about 2g/kg diet.

Homeostasis is achieved largely by a balance between gut absorption and renal excretion. Adrenal, thyroid and parathyroid hormones influence status, although plasma $[Mg^{2+}]$ has a less potent effect on PTH than does $[Ca^{2+}]$. PTH causes an increase in plasma Mg by increasing absorption from intestines and renal tubules and resorption from bone (PTH also requires Mg ions for activation of adenylate cyclase in bone and kidney). Aldosterone secretion (see Sodium, this chapter) causes a lowering of plasma Mg and an increase in urinary Mg excretion. Typical diets may not meet the horse's Mg need without supplementation.

Table 3.4 Daily requirements of Mg (g).

	Mature weight (kg)	
	400	500
Adult		
Rest	5.6	7.0
Medium	6.5	8.0
Last 90 days of gestation	6.5	8.0
Peak lactation	6.6	8.1
Growing: age (months)		
3	4.2–5.5	4.8–6.8
6	4.0–5.2	4.4–6.3
12	4.2–5.3	4.5–6.5
18	4.5–5.6	5.0–7.0

A rarely observed frank dietary deficiency of Mg leads to hypomagnesaemia associated with loss of appetite, nervousness, sweating, muscular tremors, rapid breathing (hyperpnoea), convulsions, heart and skeletal muscle degeneration and, in chronic cases, mineralization of the pulmonary artery caused by deposition of Ca and P salts. Normal blood serum values are given in Table 3.1.

Mg of vegetable origin, naturally present in feed, is available in proportions ranging from 45% to 60%, the more digestible sources being milk and possibly lucerne. Absorption of Mg from high-temperature-dried lucerne is greater than it is from timothy hay, according to evidence from Edinburgh (see also ‘Oxalates and other dietary factors affecting Ca absorption’ this chapter). Sugar beet pulp and beet molasses are also reasonably good sources of digestible Mg (both contain about 2.8 g Mg/kg DM). Large amounts of dietary P seem to depress absorption of Mg slightly, but not as effectively as do dietary oxalates. Bacterial phytase in the gut may assist absorption. The inorganic sources of Mg – Mg oxide (calcined magnesite), sulphate and carbonate – are all about 70% absorbed in the horse, although oxides from different countries of origin have differing availabilities. Generally, therefore, Mg carbonate is a more reliable source. An increase in the dietary level of Mg from 1.6 to 8.6 g/kg was shown to increase Ca absorption without an effect on P. Grace *et al.* (1998b) measured the mineral composition of horses aged 150 days (approximately 190 kg BW) and, assuming a growth rate of 1.00 kg/day on pasture, estimated the daily requirement of these horses for Mg to be 4.4 g/day. Table 3.4 gives the daily requirements of Mg for horses weighing 400 and 500 kg.

Potassium (K)

Potassium is subject primarily to precaecal absorption, where 52–74% is absorbed. An intake of 46 mg K/kg BW/day during rest is adequate for a positive K balance in adult horses. Foals require more, perhaps as much as 150–200 mg/kg BW, that is, 7 g

Table 3.5 Serum total concentrations (mmol/l) of electrolytes in TB foals (Sato *et al.* 1978).

Days from birth	Ca	P	Na	K	Cl	Mg
0	2.64	1.51	131.6	4.68	103.6	0.88
10	2.62	1.48	142.0	4.42	97.8	0.97
20	2.83	1.79	138.6	4.08	98.4	0.90
50	2.34	2.11	137.2	4.28	96.6	1.32*
90	2.50	1.74	140.0	3.96	99.8	—
120	2.23	1.63	136.4	4.12	101.4	—

*Thirty days of age.

K/kg diet. Losses during sweating or diarrhoea increase the need considerably. Young foals may become deficient in K as a result of persistent diarrhoea and this in turn tends to precipitate acidosis. Spontaneous changes in plasma $[K^+]$ can therefore result from strenuous exercise; this will be discussed in Chapter 9. The normal plasma K concentration is in the range 2.4–4.7 mmol/l (or mEq/l) (Table 3.5). Nevertheless, the plasma concentration increases during episodes of acidosis, as intracellular, red cell K (normal concentration 83–100 mmol/l) (Muylle *et al.* 1984b) exchanges for H^+ ions, when cardiac arrhythmias can occur. Red cell K concentrations <81 mmol/l are accompanied by signs of skeletal muscle weakness (Frape 1984b). The means of assessing K status and the major causes of K depletion and their therapy are discussed in Chapter 11.

Deficiency

A dietary deficiency of K may reduce appetite and depress growth rate; a reduction in plasma K (hypokalaemia) occurs and in extreme deficiency there may be clinical muscular dystrophy and stiffness of the joints. Hypokalaemia can occasionally result from persistent diarrhoea, or from excessive sodium bicarbonate administration. In depletion of ponies, the K losses in urine did not fall below 20 mmol/l (Meyer *et al.* 1986) and the K content of sweat remained invariable at about 27 mmol/l (cf. sodium). Blood plasma K decreased from 3.5 to 2.3 mmol/l, whereas red cell K changed little. However, the K depletion of the skeleton was as much as 60%, whereas losses in muscles, vital organs and GI contents were only 9%, 15% and 7%, respectively. Food and water intake decreased, the ponies were more excitable and exercise exhaustion occurred sooner.

Hyperkalaemic periodic paralysis

A syndrome of episodic weakness in horses, accompanied by elevated serum K concentrations – hyperkalaemic periodic paralysis (HPP) – has been described in horses (Naylor *et al.* 1993) and it is apparently confined to descendants of the American Quarter Horse Impressive. The genetic cause has been identified and

progress is being made to eliminate the disease (Meyer *et al.* 1999). HPP is not caused by a dietary K deficiency. It is accompanied by myotonia, facial muscle spasm and fasciculations and recumbency. Tracheotomy may be required if there is severe dyspnoea. Most episodes resolve spontaneously in 15–120 min, but may require parenteral administration of Ca gluconate, Na bicarbonate, dextrose, insulin and subsequently dietary management. This situation reflects the exchange of intracellular K^+ with H^+ in acidosis mentioned in Potassium, above.

Sources

Cereals are relatively poor sources of K, but hay contains 15–25 g K/kg; thus, most diets should contain ample if at least one-third is in the form of good-quality roughage. Animals in heavy work generally consume more cereals, thus lowering dietary K when losses in sweat would normally be increasing. Lush pastures can contain large amounts of K in the dry matter and so theoretically may interfere with Mg metabolism.

Sodium (Na)

Sodium (Na) is the principal determinant of the osmolarity of extracellular fluid and consequently of the volume of that fluid. Chloride concentration in the extracellular fluid is directly related to that of Na. Na is derived from ingested food and its excretion via the kidney is controlled by the renin–angiotensin–aldosterone system. Hyponatraemia can occur from reduced intake of Na and it causes aldosterone secretion; however, Na deficits can also occur through excessive losses from the GI or urinary tract relative to water loss. These may indicate intestinal obstruction, enterocolitis or renal failure. Excessive sweating normally causes hypernatraemia. In some other species, increased plasma levels of aldosterone result in increased urinary Mg excretion, although what occurs in the horse is unclear.

Na is reabsorbed by the large intestine to the extent of 95%. In Na deficiency, the reabsorption may reach 99% and renal Na losses are reduced, thus conserving the tissue content, according to Meyer's group in Hanover (Meyer *et al.* 1982b). The resting horse, receiving a Na deficient diet, can conserve Na. The Na content of the sweat of working horses is, however, decreased only slightly, being partly replaced by K. Such Na depleted horses exhibit a licking habit and a craving for large amounts of sodium chloride, decreased cutaneous turgor, reduced feed intake and ultimately cessation of eating, muscular and nervous dysfunction (muscular tremor, gait and chewing incoordination). In advanced Na depletion, plasma Na and chloride concentrations fall to 120 and 70 mmol/l, respectively, with an increase in plasma K of up to 5.5 mmol/l and a decrease in total body water, largely through increased dry matter of the GI tract contents.

Although pasture grass may contain as much as 18 times more K than Na, supplementary Na in the form of common salt for grazing stock is normally unnecessary. Forages are a richer source of Na than are cereals and within normal

Endogenous losses (faecal + renal + cutaneous)	3 + 2 + 1
Cutaneous maintenance losses by perspiration	14
Maintenance, minimum	20
Maintenance, recommendation	80
Maintenance + exercise, 5, 20 or 50 g sweat/kg BW	108, 190 or 355
Maintenance + pregnancy up to 90 days ante partum	80
' ' + ' ' last 90 days of pregnancy	82
Maintenance + lactation, 3rd month	89–93
Maintenance + growth until 6th month	93
Maintenance + growth, sixth to twelfth months	85

Chloride (Cl)

TRACE ELEMENTS

Most stabled horses in the UK now receive supplements containing variable quantities of trace elements, and the horse seems able to cope with some measure of abnormal intake without showing clinical signs of toxicity or deficiency. Those trace elements of prime importance in the diet of horses are discussed here. Cobalt (Co) is considered in Chapter 4 under vitamin B₁₂. The variety of geological strata underlying UK soils yields grazing areas that cause clinically recognizable signs of specific deficiencies in cattle and sheep. There is biochemical evidence to show that horses and ponies in these areas may similarly reflect their nutritional environment. Tables 3.7 and 3.8 give average serum values for some trace elements. Abnormalities in leg growth and development of foals and yearlings have been reported to be

Table 3.7 Values for four trace elements ($\mu\text{mol/l}$) in normal serum or plasma and milk of mares (Smith *et al.* 1975; Blackmore & Brobst 1981; Cape & Hintz 1982; Schryver *et al.* 1986; Lawrence *et al.* 1987b; Bridges and Harris 1988; Saastamoinen *et al.* 1990) (also see Chapter 12 for Cu determinations).

	Blood	Milk		
	Serum or plasma	Partum	1–8 days	9–120 days
<i>Cu</i>				
TB, USA mainly stabled	24–35	16	10–12	4–6
TB	8–18			3 ¹
TB, 7, 14, 28 days of age	6, 14, 25			
Quarter Horse	5–31			
Horse/pony, gestation ²	16.3			
Horse/pony, lactation ²	14.7			3.8
Horse/pony, yearlings	15.3–26.1			
Horse/pony, 2–3 years old	21.3			
<i>Fe</i>				
TB	28 $\pm$ 7			
Quarter Horse	28			
Arabian	23	24	17	12
Standardbred	30 $\pm$ 7			
Shetland	19			
<i>Zn</i>				
TB, USA, stabled	—		46	60
TB, UK, pastured	17 $\pm$ 7	98	52	36
TB, UK, stabled	26 $\pm$ 8			
TB, 7, 14, 28 days of age	17, 12, 12			
Horse/pony, gestation ²	5.8			
Horse/pony, lactation ²	7.6			31.3
Horse/pony, yearlings	10.7, 12.5			
Horse/pony, 2–3 years old	10.1			
<i>Pb</i>				
TB and Standardbred:				
suckling to adult (mean)	0.8–1.9			
mixed breeding	7–8			

¹ Unpublished data of author.

² (Saastamoinen *et al.* 1990). Finnhorse breed.

associated with dietary deficiencies of copper (Cu), manganese (Mn) and selenium (Se), and toxicities of iodine (I) and lead (Pb).

The extent to which pasture plants extract trace metals from soil depends on the soil's pH and moisture content and the plant species. Effects may also be attributable to the root systems of plants as legumes and many herbs have deeper roots than grasses do. The levels of trace elements in herbage are clearly of importance, but the horse will also consume soil while grazing. Soil intake will depend on the density of the herbage. In certain conditions cattle and sheep may consume more than 10% of their daily intake of dry matter as soil.

Table 3.8 Normal blood characteristics of horses.¹ Ranges indicate approximately ± 1 SD from mean.

Sample	Characteristic	Units per litre	Yearlings	Horses in training	Mares	Foals
Plasma	Albumin	g	27–28	34	27	25
Serum	AST	iu	140	150–400	140	70–120
Serum	CK	iu	43	50–70	43	53–57
Serum	SAP	iu	100–120	85–95	70–80	150–400
Serum	ALT	iu	1–6.7	1–6.7	1–6.7	NA
Serum	GGT	iu	18–30	20–30	18–19	13–16
Red cell	GSH-Px	u/ml ⁷	15–25	15–25	—	—
Serum	LDH	iu	45–100	45–100	—	—
Serum	SDH	iu	0.8–1.2	0.8–1.2	—	—
Serum	Bilirubin, total ²	μ mol	34	34–39	26	38–55
Serum	Fasting glucose	mmol	3.5–5	3.5–5	3.5–5	3.1–4.2
Serum	T ₄	nmol	5–39	5–35	3–56	10–150
Serum	T ₃	nmol	1.5–2	1–2	0.9–1.4	2–7
Serum	Creatinine	mmol	140	170–185	140	150–190
Serum	Cu	μ mol – stable	15.2 ³	12.4	15 ⁴	6 to 20 ⁵
Serum	Cu	μ mol – grass	—	15.9	16–26	20 ⁸
Serum	I	μ mol	—	—	0.6	0.8–0.85
Serum	Zn	μ mol – stable	—	26	9–12	13
Serum	Zn	μ mol – grass	—	17	9–12	—
Serum	Mo	μ mol	—	0.31	—	—
Serum	Se	μ mol	0.8–1.3	1.5 ⁶	0.8–1.2	0.6–1.1
Whole blood	Se	μ mol	—	1.6 ⁶ –3.0	0.8–1.1	0.5–0.7
Plasma	α -Tocopherol	mg	1–3	1.5–4.4	1.8–2.6	1.1–2.4
Plasma	α -Tocopherol	μ mol	2.3–6.8	3.4–10	4.1–5.9	2.5–5.5
Plasma	Retinol	μ g	180	180	150–300	150
Serum	25-Hydroxy vitamin D	μ g	—	—	2.9–3.6	2.2–2.5
Whole blood	Cyanocobalamin	μ g – stable	3.7–6.6	1.2–6.6	3.7–6.6	3.7–6.6
Whole blood	Cyanocobalamin	μ g – grass	2.8–20	2.8–20	2.8–20	2.8–20
Serum	Folate	μ g – stable	4.5–12	4.5–12	4.5–12	4.5–12
Serum	Folate	μ g – grass	5.3–13.5	5.3–13.5	5.3–13.5	5.3–13.5
Whole blood	Thiamin	μ g	28	30	33	24
Plasma	Ascorbic acid	mg	—	2.5–4.5	—	—

¹ Data are drawn mainly from TBs. See Table 3.1 for serum Ca, P, Mg, Na, K and Cl values.² Mainly unconjugated.³ Ponies.⁴ Whole blood.⁵ Rising from 1 week of age to plateau at 4 weeks.⁶ Minimum adequate.⁷ One enzyme unit of GSH-Px activity = 1 μ mol NADPH oxidized/min.⁸ Plasma.

Abbreviations: ALT, alanine amino-transferase; AST, aspartate amino-transferase; CK, creatine lactate; GGT, γ -glutamyl-transferase; GSH-Px, glutathione peroxidase; iu, international unit; LDH, lactate dehydrogenase; SAP, serum alkaline phosphatase; SDH, sorbitol dehydrogenase; T₃, triiodothyronine; T₄, thyroxine.

Mixed supplements

Ott & Asquith (1995) measured the response of growing TB and Quarter Horses from 340 to 452 days of age to trace mineral supplements, when given concentrates to appetite and coastal Bermuda grass (*Cynodon dactylon*) hay at the rate of

1 kg/100 kg BW daily. The unsupplemented total diet contained 195 mg Fe, 36 mg Mn, 41 mg Zn and 4.8 mg Cu, each per kg DM. The supplements had no influence on growth and development, but mineral deposition in the third metacarpal was increased by the trace mineral mix, although not by Cu or by Cu plus Zn, excluding the other elements. The results suggested that the other trace minerals (Fe, Mn, Co or I) were more critical. However, previous work by the same group (Ott & Asquith 1989) indicated that bone mineral deposition in yearlings was increased when a trace mineral mix containing Fe, Mn, Zn and Cu was added to a natural diet containing lower concentrations of these elements than those recommended by the National Research Council (NRC) (1978).

Mineral content of horse hair

The mineral content of animal hair varies not only with mineral intake, but also with season, breed, age, hair colour and body condition. Nickel (Ni) intake is apparently correlated with Ni in hair, but other heavy metals, at least at subtoxic intakes, are not (toxic intakes of Pb cause elevated hair Pb). There is little simple relationship between minerals of nutritional importance and their concentration in horse hair.

Copper (Cu) and molybdenum (Mo)

Hypocupraemia occurs widely in grazing cattle, attributed frequently to an excess of molybdenum derived from the underlying Mo-rich strata, particularly Lias clays and marine black shales of Jurassic and Carboniferous ages. The high levels of Mo associated with relatively low copper lead to Cu:Mo ratios in the herbage narrower than 6:1, causing Cu deficiency on the so-called teart pastures in these areas. Hypocupraemia can also occur, owing to low Cu levels per se in the soils and herbage.

The horse is not as susceptible to clinical signs of Cu deficiency as are ruminants, but signs have been described, such as erosion of the articular cartilage of joints, and anaemia and haemorrhage in parturient mares. Moreover, Mo and sulphate do not have the same impact on Cu status in horses as they have in cattle. Thiomolybdates, which bind Cu (CuMoS_4 is very insoluble), have not been detected in horses with dietary Mo concentrations of 10 mg/kg. Their formation may depend on the presence of a rumen and its microbial activity, that is, an extensive microbial activity in a region of the GI tract proximal to absorption sites for Cu. Thus, Rieker *et al.* (1999, 2000) have shown that a concentrate containing 5–20 mg Mo/kg given to geldings with grass hay (50:50) had no adverse effect on the absorption and retention of Cu.

Measurement of Cu status and dietary requirements

Excessive intakes of Cu are hazardous to sheep and, to a lesser extent, to cattle. Ruminants differ from nonruminants in the propensity of the former to store Cu in their livers at low dietary Cu intakes. On the other hand, very high dietary Cu levels (>4 mmol/kg DM) are required to increase liver Cu of ponies substantially (referred

to by Suttle *et al.* 1996). (Note: any increase above the minimum dietary requirement seems to lead only to some additional liver Cu storage.) For this reason horses are less subject to intoxication by dietary Cu. There is a curvilinear relationship in ruminants between liver Cu and plasma Cu concentrations. In ruminant plasma, or serum, Cu values plateau and, excepting toxic crises, they rarely exceed 16 µmol/l, even with the liver at >800 µmol/kg fresh weight. In 44 horses, Suttle *et al.* (1996) found a linear relationship:

$$Y = 7.00811 + 0.086019X \text{ (R}^2, 0.168\text{)}$$

where Y is µmol Cu/l serum, X is µmol Cu/kg fresh liver (values were restricted to liver Cu <190 and serum Cu <29) and R² is the proportion of the variation, or sum of squares, attributable to the model (in this case a value of 0.168, i.e. 16.8%, of the variation is accounted for by the relationship that the equation describes). In 48 horses, Suttle *et al.* (1996) found mean Cu concentrations of:

- 16.7 µmol/l serum, with 13.5 and 19.5 µmol/l as lower and upper quartile values;
- 113.7 µmol/kg fresh liver;
- 172.5 µmol/kg feed (11 mg/kg) with 61.1 and 233.8 µmol/kg as lower and upper quartile values.

The animal has a need to maintain a normal cellular Cu concentration, measurable as hepatic concentration. In the horse, Suttle *et al.* (1996) concluded that adequate equine liver levels would be achieved by a dietary concentration of 20 mg/kg DM and that these liver and dietary concentrations correspond to 16 µmol/l serum. These authors propose 16 µmol/l as a threshold value to distinguish the normal from subnormal serum values and a serum level of 11.5 µmol/l to distinguish deficient from marginal liver Cu concentrations of 52.5 µmol/kg fresh weight. Unfortunately, as Suttle *et al.* point out, high serum Cu values occur following inflammation, infection or vaccination and liver samples are then preferable, but not readily available. (Plasma Cu may be unreliable as a guide to Cu status, as about 70% of the circulating Cu in the horse is present in the form of caeruloplasmin [EC 1.16.3.1], an acute phase protein.) Therefore an alternative cellular source may be preferred as a means of assessing Cu status.

Cu deficiency decreases the activity of Cu–Zn superoxide dismutase (EC 1.15.1.1) of leucocytes and platelets. The measurement of this activity is not easy. The author's colleagues (Williams *et al.* 1995) have routinely used mononucleated leukocyte and platelet Cu contents and platelet cytochrome c oxidase (EC 1.9.3.1) activities as guides to status. Mononuclear cell Cu shows good promise (Table 3.9). The Cu contents will be present mainly as enzyme cofactor Cu (see Chapter 12).

Cu is transferred to the foetal liver, which, like the neonatal liver, contains more Cu than that of older foals, or of their dams. The low serum Cu concentration of neonatal foals does not rise to normal adult levels until about 28 days of age (Kavazis *et al.* 2001). Experiments in New Zealand have shown that when the dietary Cu of the grazing pregnant mare is increased from approximately 6–30 mg

Table 3.9 Range in leukocyte Cu and Zn contents in seven Shetland ponies receiving poor hay only, before and after supplementation with Cu and Zn for 50 days (author's unpublished data).

	Cu ($\mu\text{g } 10^{-9}$) in leukocyte cells	Zn ($\mu\text{g } 10^{-9}$) in leukocyte cells
Before supplementation	0.11–0.18	2.57–6.25
After supplementation	0.40–2.86	2.57–10.57

Cu/kg DM throughout the last four to five months of gestation, then the Cu content of the foal's liver at birth can be increased by two-thirds. Pearce *et al.* (1998a) studied grazing mares and their foals, during the second half of gestation, raised on a pasture that contained 4.4–8.6 mg Cu/kg DM. Half the mares were supplemented with 0.5 mg Cu/kg BW daily. The foals from control and supplemented mares were either retained as controls or supplemented at 0.2 mg/kg rising to 0.5 mg/kg BW at 49 days of age. Plasma Cu and caeruloplasmin concentrations in the foals were unaffected, whereas liver Cu concentration increased, with supplementation. Mare supplementation alone (Pearce *et al.* 1998b) decreased radiographic indices of physitis in the distal third metatarsal bone and the prevalence of articular cartilage lesions at 150 days of age, without evidence of effect on clinical developmental orthopaedic disease (DOD).

Mare's milk may provide less Cu than the suckling foal requires daily (Cu content of milk is about $3 \mu\text{mol/l}$ (0.19 mg/l), whereas pasture grass and grass hay may contain 4–9 and 10–12 mg Cu/kg DM respectively. Therefore, with the object of increasing the Cu and Zn contents of the milk, and decreasing the risk of osteochondritis dissecans (OCD), Baucus *et al.* (1987) doubled the Cu and Zn contents of the mare's diet to 53 mg Zn/kg and 12 mg Cu/kg at parturition. Nevertheless, the Cu and Zn contents of the milk were unaffected.

The suckling foal requires a source of Cu to tide it over the period during which it eats little grass and dry food. These needs are normally met when the foetal liver stores about $300 \mu\text{g}$ Cu/g liver DM, or more. The stores should range between 300 and $600 \mu\text{g/g}$ when the pregnant mare has received adequate Cu in her diet. Although Hoyt *et al.* (1995b) found that miniature horses absorbed 42.2–50.7% of the total Cu from diets containing 12 mg/kg, Ott & Asquith (1994) reported that serum Cu and Zn concentrations in foals were increased only when their mothers received chelated trace minerals, rather than inorganic sources, during pregnancy.

The obligatory losses of Cu in the faeces of ponies amount to about 3.5 mg/100 kg BW daily in the presence of low levels of dietary Mo (Cymbaluk *et al.* 1981a,b). However, in order to allow for adverse interactions with other trace elements and to maximize iron (Fe) retention, a dietary intake of 15–20 mg/kg dry feed is recommended for growing horses. Foals need 25–30 mg/kg feed to reduce, but not

eliminate, risk of cartilage erosion. Grace *et al.* (1998b) measured the mineral composition of horses aged 150 days (approximately 190kg BW) and, assuming a growth rate of 1.0kg/day on pasture, estimated the daily requirement for Cu to be 26mg/day.

Cu and cartilage formation

(See also 'Growth of foals', Chapter 8). The bones and cartilage of Cu-deficient animals show increased defects and fragility, and contain an enhanced proportion of soluble collagen. This solubility is caused by a reduction in the cross-linking within the molecules of collagen and elastin that require Cu as the cofactor of lysyl oxidase (EC 2.3.2.3). Osteoblast function is inhibited by Cu deficiency, whereas osteoclast function is unaffected. Excess dietary Cu can interfere with bone metabolism causing inhibition of collagen synthesis and a loss of bone density. Amongst 629 Hanoverian warmblood foals in Germany, 222 presented with osteochondrotic lesions not correlated with growth rate or with protein, Ca, P, Cu, Zn or Se intakes (Coenen *et al.* 2003a; Vervuert *et al.* 2003a), although the dietary levels of these nutrients did not meet recommendations (GEH 1994). Preliminary evidence of Firth (1998) indicated that Cu supplementation of the foal may reduce bone mineral density of the radius, third metacarpal and third carpal. Moreover, neither organic nor inorganic supplements of Cu and Zn influenced blood carboxy-terminal propeptide of type I procollagen, or bone density, in exercised yearling geldings given a basal diet meeting NRC (1989) recommendations (Baker *et al.* 2003).

On the other hand, *in vitro* studies indicated that, in a dose-dependent manner, copper reversed proteoglycan depletion of cartilage culture in the presence of synovial fluid, so that Cu had a *chondroprotective* effect (Davies 1998). Davies hypothesised that Cu exerts an anti-inflammatory action.

Van Weeren *et al.* (2003) found no relationship between foal, or mare, liver copper concentration and osteochondrosis status at 5 or 11 months. However, as the foals increased in age from birth, the expected decrease in number and severity of osteochondrotic lesions was less where Cu status was low at birth than it was for foals with a high Cu status at birth. So there may be an effect of high Cu status on the natural process of repair of early lesions, indicating the preeminence of the Cu status of the pregnant mare.

An excess of dietary Cu of up to 791 mg/kg diet in ponies for six months (Smith *et al.* 1975) led to liver Cu concentrations of over 4000mg/kg DM, apparently causing no liver damage and no adverse effect on fertility or other characteristic.

Zinc (Zn)

A deficiency of dietary zinc in many domesticated animals, including the horse, depresses appetite and growth rate in the young, causes skin lesions and is associated with a depression of Zn concentrations in the blood. A deficiency of Zn in the rat and several other species causes abnormal development of ribs and vertebrae,

cleft palate, micrognathia (undersized mandible) and agenesis (absence) of long bones, but there is little direct evidence of this in the horse. Excess Zn may exacerbate bony lesions induced by low Cu diets.

Siciliano *et al.* (2003b) observed positive total immunoglobulin and IgM humoral immune responses in weanling horses from a supplement of Cu, Zn and Mn. Zinc has a potent immunomodulatory capacity, particularly influencing T-helper-cell organization and cytokine secretion. *Culicoides* hypersensitivity in horses, causing a pruritic skin disease, resembles early and late phase of type I hypersensitivity reactions in man. Stark *et al.* (2001) determined that the severity of *Culicoides* hypersensitivity in Icelandic horses expressed a low, but significant, negative correlation with plasma zinc concentration. However, their more recent observations indicate that *Culicoides* bites are a cause of Zn redistribution between plasma and blood cells, and as plasma holds only 10–23% of total blood Zn, plasma Zn level is not a reliable indicator of nutritional status. Thus, the depression in plasma zinc is quite likely to be a reaction to the bite of this fly.

Zn supplements are claimed to benefit hoof wall development, although Siciliano *et al.* (2001) could not influence the trace mineral content of hoof wall, or wall growth rate, hardness and tensile strength, with supplements of Cu, Zn or Mn, given to light mares as inorganic salts, or as amino acid chelates. More recently Siciliano *et al.* (2003a) increased hoof wall hardness, but not other characteristics of the wall, with similar supplements of Cu, Zn and Mn.

Zn as enzyme cofactor

Zn is a cofactor for over 200 enzymes in animals, either as part of the molecule or as an activating cofactor. The enzymes include alkaline phosphatase (EC 3.1.3.1), collagenase (EC 3.4.24.3) and carbonic anhydrase (EC 4.2.1.1), all required in bone formation. Alkaline phosphatase also requires Mg, and excess dietary Zn may inhibit the enzyme if Mg is displaced. A deficiency of Zn thus has fairly widespread physiological effects, but quite high dietary levels are required for toxicity.

An increase in dietary Zn from 26 to 100 mg/kg progressively increases serum Zn concentrations. The dietary requirement of the horse is approximately 50 mg/kg dietary DM. Grace *et al.* (1998b) measured the mineral composition of horses aged 150 days (approximately 190 kg BW) and, assuming a growth rate of 1.0 kg/day on pasture, they estimated the daily requirement for Zn to be 152 mg. Hudson *et al.* (2001) measured the true digestibility and requirements for Cu, Zn and Mn from a mixture of organic and inorganic sources in sedentary and exercised horses. They found a Zn requirement of 461 mg/day in exercised horses, cf. sedentary horses of 274 mg/day (Table 3.10). Supplements normally used include zinc carbonate or sulphate. These inorganic salts possess a higher availability than do phytate salts of zinc in cereal grains and oilseed meals. The efficiency of Zn absorption in all forms is probably affected more by diets rich in phytate than is the absorption of other trace elements, but, even so, high phytate concentrations are unlikely to depress the utilization of Zn by more than 30–40%.

Table 3.10 The true digestibility, endogenous loss and estimated daily requirements for copper, zinc and manganese in sedentary (SED) and exercised (EX) TB geldings of 11 years of age and 534 kg BW (Hudson *et al.* 2001).

		Cu	Zn	Mn
Digestibility %	SED	41.8	25.4	57.9
	EX	54.5	14.3	40.2
Endogenous loss, mg/day	SED	15.7	65.2	304.8
	EX	20.3	69.6	163.6
Requirements, mg/day	SED	44.2	274.4	528.6
	EX	35.0	461.3	408.3

Zn is one of the less toxic of the essential trace metals, yet where there is industrial pollution of pastures, grazing animals may show signs of toxicity. Toxic dietary concentrations probably exceed 1000 mg/kg. Zinc intoxication is associated with reduced Ca absorption. A dietary level of 5.4 g Zn/kg causes anaemia, epiphyseal swelling, stiffness and lameness, including breaks in the skin around the hooves (Willoughby *et al.* 1972a,b).

In comparisons of the efficacy of organic and inorganic sources of Cu and Zn, two out of three studies indicated more effective use of organic sources in young horses (Miller *et al.* 2003; Siciliano *et al.* 2003b), but the third in adult horses indicated low absorption efficiencies of both sources (Wagner *et al.* 2003).

Cu and Zn interactions

In man and most other domestic animals that have been investigated there is an antagonism between Zn and Cu. Excessive intakes of Zn, especially where dietary phytate and Ca are not abundant, can cause Cu deficiency if the dietary Cu level is marginal. Investigations in horses have not demonstrated similar effects. Hoyt *et al.* (1995b) found that dietary Zn concentrations of 73–580 mg/kg, provided as zinc oxide, had no influence on either Cu absorption or retention by horses from diets containing 12 mg Cu/kg. However, as little as 100 mg Zn/kg diet for horses has been shown to increase the faecal loss of Cu and to lower blood Cu by about 10%. This change in blood concentration may not itself reflect any change in Cu absorption efficiency. Nevertheless, as Cu adequacy seems to be critical for proper bone development we should assume that dietary Zn at concentrations above 100 mg/kg are to be avoided.

Amino acid chelation of trace elements avoids any interruption to Zn absorption by other metals and the competition for absorption is then between the amino acids. Experiments have shown that chelated mixtures of Mn, Zn and Cu, cf. inorganic mixtures, have increased the growth rate of hoof horn in yearlings given a pelleted concentrate containing 120 g protein/kg at the rate of 1 kg/100 kg BW with hay.

Experiments with chelated Zn and Cu given to pregnant mares have led to an increase in plasma concentrations of Zn and Cu in their foals. It may be concluded that the trace element allowances for the pregnant mare are more important than those given during lactation for the welfare of the foal.

Manganese (Mn)

Manganese is required as a cofactor for glycosyltransferases, which catalyse the transfer of a sugar from a nucleotide-diphosphate to an acceptor, and so Mn is essential in several stages of glycosaminoglycan–chondroitin sulphate formation. Thus, epiphyseal cartilage and bone matrix formation are compromised by a deficiency of Mn. Mn is also required as a cofactor in Mn-containing superoxide dismutase.

A deficiency of Mn is thought to be a cause of enlarged hocks, and, by affecting the growth plate, to shorten legs with characteristic knuckling-over of joints. In the USA, excessively high concentrations of Ca in some samples of alfalfa have been said to precipitate a flexural deformity of the legs of growing horses that was rectified by Mn supplements. The young also seem to suffer lameness and incoordination of movement if they lack sufficient Mn, and Mn deficiency is a possible explanation of tiptoeing in situations where suckled foals are on pasture containing <20mg/kg DM. A severe deficiency can give rise to resorption *in utero*, or death at birth, and lesser deficiencies may provoke irregular oestrous cycles. Lawrence *et al.* (1987b) reported normal plasma Mn values in yearling ponies of 100–180µmol/l serum.

The true digestibility of dietary Mn is approximately 50% (Hudson *et al.* 2001; Table 3.10). It was shown by Sobota *et al.* (2001) that a total dietary Mn concentration of 36mg/kg (using a concentrate containing 23 or 131 mg/kg and coastal Bermuda grass hay of 55mg/kg) is inadequate in yearling TB and Quarter Horses for maximum weight gain, long bone growth and biochemical indices of bone formation. The evidence of Hudson *et al.* (2001) indicates a requirement of approximately 50mg/kg DM in adult horses.

Iron (Fe)

Most natural feeds, apart from milk, are fairly rich sources of iron, even when the availability may be questionable, and deficiencies are unlikely unless the horse is anaemic through heavy parasitization. The foal is born with an adequate store of liver Fe and the foal's grazing activity is normally an adequate supplement to the mare's milk, which contains meagre amounts of most trace elements, with the possible exception of selenium (Se) in mares supplemented with this element. The levels of Fe in Arabian mare's milk are shown in Table 3.7.

A deficiency of Fe causes anaemia, but evidence of dietary Fe deficiency anaemia in horses is rare. A dietary concentration of 50mg/kg DM should be adequate for growing foals. Only 40mg/kg is said to meet the maintenance requirements of

adults. The basal diet used by Lawrence *et al.* (1987b) contained 436 mg Fe/kg (typical of many natural ingredient diets) and was composed of coastal Bermuda hay, maize, soya, alfalfa and minerals, including a supplement providing 40 mg Fe/kg basal diet. The addition to it of 500 or 1000 mg Fe/kg, as citrate, brought no benefit.

True dietary Fe deficiency may be confused with pseudo-Fe deficiency. Anaemia is not synonymous with Fe deficiency. Haemoglobin (Hb), which contains 67% of Fe stores in the horse, is synthesized preferentially to that of nonhaemoglobin Fe-containing proteins in Fe-depleted animals. On the other hand, blood packed cell volume (PCV) and Hb may be decreased by parasitism. Loss of red cells also occurs during TB exercise when some haemolysis of these cells occurs, and 0.6% of dietary Fe can be found in sweat (Inoue *et al.* 2003). In this situation the apparent absorption efficiency of Fe is increased, preventing Fe deficiency.

Fe status is assessed most reliably by serum ferritin concentration. Total serum Fe is an unreliable measure of status and may decline during microbial invasion, tissue injury, immunological reactions and in any inflammatory process causing an acute phase response, initiated by interleukin-1. Normal ferritin values fall between 100 and 200 µg/l serum. Deficiency may be assumed when values fall below 50 µg/l, and Fe overload may exist when values exceed 400–450 µg/l.

Adverse effects of Fe supplements

Fe-containing haematinics are widely used in the horse industry. Dietary Fe supplements and injectable Fe are used in the mistaken belief that PCV and Hb concentration can be regularly increased. The risk of Fe toxicity is a real one and antagonistic interactions with other trace elements may occur. Large, but natural, intakes of Fe in cattle can cause Cu deficiency. An induced Cu deficiency of this kind seems to be less likely in horses, although reductions of splenic Cu and Mn, associated with intakes of 1400 mg Fe/kg diet, were observed by Lawrence *et al.* (1987b). These workers also observed depressed serum and liver Zn with dietary Fe intakes of only 890 mg/kg. Interactions of this kind may arise from competition for binding by intestinal transferrin and/or other transport mechanisms.

Most Fe is stored in the liver and spleen, with no mechanism for disposal of excess, and hence excess can cause hepatitis and other forms of liver damage. The toxic level depends on several factors, including concurrent disease processes, previous liver damage and vitamin E and Se status. Fe is involved in oxidation–reduction reactions. When glutathione peroxidase activity is depressed and vitamin E status is reduced and there is an Fe overload, catalase activity increases, with consequent liver damage, coagulopathy and raised mortality. Fe overload in the spleen may be the cause of the lymphopenia frequently observed in neonatal cases.

Neonatal foals are particularly susceptible to iatrogenic Fe toxicity. Ferrous-Fe (divalent) is more soluble than ferric-Fe (trivalent) and therefore ferrous sulphate or fumarate is generally used in supplements and they are more likely to be a cause

of toxicity. Ingestion of a single large dose of ferrous fumarate, shortly after birth, can cause death within five days. Daily administration of as little as 300 mg Fe in the ferrous form has been associated with signs of Fe toxicity in adult TBs. In extended Fe intoxication there is frequently:

- hepatic dysfunction, expressed as lethargy;
- yellow discolouration and petechial haemorrhages of mucous membranes; and
- thrombocytopenia and elevated activities of both serum GGT (EC 2.3.2.2) and alkaline phosphatase (ALP).

These enzyme changes are indicative of cholestatic hepatopathy, with periportal bile ductule proliferation, as one origin of these enzymes is biliary epithelia.

Fluorine (F)

Fluorine readily substitutes for the hydroxyl ion in bone and teeth hydroxyapatite, creating a more stable crystal of Ca, P and Mg. It does not diffuse into formed bone, but becomes incorporated during bone formation. F increases osteoblast number by increasing osteoprogenitor cell proliferation. Risks of dietary excess (fluorosis) in grazing horses probably exceed risks of deficiency, as F has a narrow therapeutic index. Excess contamination of pastures, especially from brickworks, causes a softening, thickening and weakening of bones, through defects in mineralization, which are probably not prevented by Ca and vitamin D. A decrease in industrial effluent in many countries has ensured that very few cases occur today. The horse seems to excrete more F in its faeces than do cattle, but dietary concentrations should not exceed 50 mg/kg. A world shortage of sources of digestible P has led to an increase in the use of rock phosphates; as some of these are rich in F a careful scrutiny of their composition is essential before purchase and use.

Iodine (I)

Iodine is a relatively rare element in the earth's crust and it does not seem to be required by mono- and dicotyledonous plants, in which the concentration is low. In man and animals both I deficiency and toxicosis generally result in hypothyroidism (toxicosis may cause hyperthyroidism in some individuals). Goitre may then occur, with hyperplasia and hypertrophy of the thyroid gland, induced by the elevated secretion of thyroid-stimulating hormone (TSH) originating in the anterior pituitary in the absence of feedback inhibition. This is not necessarily the outcome of I toxicosis in the pregnant mare (see below).

Insofar as the foetus is concerned, the mare is at risk throughout gestation, and maybe even shortly before fertilization, in respect of both I deficiency and toxicity. When receiving insufficient dietary I mares may show no external signs, but may exhibit abnormal oestrous cycles and then produce hypothyroid foals. Serum triiodothyronine (T_3) and thyroxine (T_4) concentrations in the mare are low, generally below 1.3 and 19 nmol/l (0.7 and 15 µg/l), respectively (Table 3.11). The I

Table 3.11 Normal ranges for plasma T₃ and T₄ concentrations in TBs (nmol/l).

	T ₃	T ₄
Pregnant mares	0.6–2	3–56
Foals*	2–10	10–150
Adults in training	0.3–2	5–28

*T₃ levels increase to a maximum at 48 h of age and then decline.

concentration of deficient mare's milk is below 20 µg/l. A deficiency with the above signs is shown when mares graze inland pasture areas that are frequently deficient in I.

Deficiency signs in foals

Deficiency signs in foals are enlarged thyroid glands, weakness, persistent hypothermia, respiratory distress and high neonatal mortality. There is an increased susceptibility to infectious disease and respiratory infections are frequent. T₄ levels in foal serum will be low, whereas T₃ concentration may be normal. Plasma T₃ and T₄ levels are generally not too helpful in diagnosis and their correlation is poor. Nevertheless, T₄ concentration is depressed in hypothyroidism. Of greater value is inspection of feed and determination of I concentrations in feed samples and in plasma.

Toxicity signs in mares and foals

In mares given 300–400 mg I daily in the feed, infertility and abortions occur. Some mares develop hyperthyroidism, expressed as elevations in the plasma concentration of both T₃ and T₄ and suppressed plasma TSH, or they may develop hypothyroidism, expressed as normal T₃ and depressed T₄, although clinically they may appear euthyroid. At birth the foal is hypothyroid, frequently with colloid goitre, variably sized thyroid follicles, containing a single layer of low cuboidal cells and there are low concentrations of circulating T₃ and T₄. Interestingly, the tracheal mucosa reveals squamous metaplasia, reminiscent of vitamin A deficiency.

In man, excess circulating iodide induces what is called the Wolff-Chaikoff block which inhibits I uptake, preventing the synthesis of T₄ from I. In many species this is a temporary phenomenon, but in the horse the block may depend on the precise level of I intake, although in the author's experience it may be only temporary. In the neonatal foal I toxicity blocks the release of T₃ and T₄ from the follicle, apparently by interfering with colloid proteolysis in the acini. Some pathologists give weight to the T₃:T₄ ratio, although in the author's experience the two hormones are poorly correlated in the healthy horse. Algae, such as red wrack seaweed, generally contain high concentrations of I and the feeding of dried kelp, a seaweed, to

pregnant mares is frequently a cause of iodism in foals, with similar clinical signs to those of an I deficiency. There have also been cases where foals given excessive thyroprotein supplements develop focal depigmentation.

If the daily I intake of a pregnant 550 kg mare is between about 40 and 400 mg I, goitre may appear in her offspring. At the upper end of the range the mare may have goitre. Intakes by the mare of 30–50 mg I daily may cause only enlargement of the thyroid in her offspring, but the foal may be euthyroid and present no other abnormality, yet hirsute foals are seen. If the daily intake exceeds about 100 mg I for any length of time during pregnancy, the newborn foal is likely to show additional signs of hypothyroidism, expressed as weakness, lethargy, high neonatal mortality, poor muscular development and, most patently, osseous dysplasia of long bones. The author's observations indicate that these include angular deformity, tendon contraction and hyperextension of the lower limbs with poor ossification of the carpal and tarsal bones. This extension causes the foal to walk on its heels with its toes raised from the ground. Abnormal growth of the bones occurs both at the epiphysis and in the shank of long bones. These bones are thin and small with cortical thickening. This thickening appears to result from a greater reduction in osteoclasia than from osteogenesis. Observations of protruding jaws (mandibular prognathism) and parrot mouth (brachygnathia) have also been recorded (Plate 3.1).

The reason for these consequences is that I is concentrated in the placenta which delivers excessive amounts to the foetus, causing congenital malformations and concentrations of I in foetal blood two to three times that of the dam's blood (yet T_3 and T_4 may be depressed). I is also concentrated in the mammary gland and is secreted in the milk. If the excessive I source given to pregnant mares is excluded from the latter's diet well before parturition the euthyroid state may be reimposed and normal foals may result. Involution of the thyroid, congenitally enlarged in young foals owing to I excess, can be brought about by removal of the I source(s). This includes removal of the mare's milk. Normal development may occur, as long as bony abnormalities have not already developed. The foal may be slightly undersized, as hypothyroidism is associated with decreased synthesis and release of growth hormone.

Goitrogens

I is required by animals for the synthesis of the hormones T_4 and the more potent T_3 . I is absorbed from the gut principally as iodides. Some feeds (raw soya beans, white clover and cabbage) contain goitrogens (see Chapter 5) which can inhibit the uptake of free I by the colloid proteins of the thyroid, preventing its incorporation into tyrosine. Thioglucosides, thiocyanates and perchlorates may be goitrogenic. The effects of these substances can be overcome by increasing the I intake. However, the goitrogenic effects of thiouracil-, thiourea- and methimazole-containing drugs cannot be overcome in this way, as they interfere with T_4 synthesis. (See 'Selenium and iodine interactions', this chapter.)



Plate 3.1 Signs of iodine intoxication at birth of foals out of mares receiving excessive amounts of dietary iodine during pregnancy. The principal signs in the stable were thyroid enlargement and bony abnormalities of neonatal foals. (a) Goitre in a foal, out of a euthyroid mare. (b) Protruding lower jaw (prognathism) of intoxicated foal. (c) Muscular weakness and long bone abnormalities of intoxicated foal. (d) Enlargement of stifle joint in intoxicated foal.

(a)



(b)



(c)



(d)

Supplements

Diets supplemented with between 0.1 and 0.2mg I/kg should adequately meet the requirements of horses. The I is preferably added as potassium iodate rather than as potassium iodide. The reason for this is that iodides are readily oxidized to I which is volatile and so gradually lost from premixes and diets; however, iodates are more toxic to handlers in concentrated form and should be handled with care.

Treatment of hypothyroidism

Young foals that are diagnosed as having goitre should not be treated with iodide, or iodate, salts in the first instance. After receiving colostrum they should be removed from their mother's milk to a source known to contain normal amounts of I. These foals are first treated with thyroxine while other analyses are undertaken to determine the cause of the malady. The author's evidence is that the thyroxine rapidly raises blood T_4 , whereas treatment with I salts has only a marginal initial influence on plasma T_4 .

Selenium (Se)

Selenium has gained prominence since the realization that it forms an integral part of the GSH-Px (EC 1.11.1.9) molecule. This enzyme catalyses peroxide detoxification in bodily tissues during which reduced glutathione (GSH) is oxidized. It is closely involved with the activity of α -tocopherol (vitamin E), which protects polyunsaturated fatty acids from peroxidation. The requirement for α -tocopherol and Se is increased in the presence of high levels of dietary polyunsaturated fatty acids – cod liver oil, linseed and corn oils – and, in fact, pasture grass. A possible case for fish oils is put in Chapter 5, although they require higher levels of protection from vitamin E, owing to a greater degree of unsaturation than of vegetable oils.

Feed fats contain variable levels of polyunsaturated fatty acids; vegetable oils are generally much richer sources of these acids than are hard animal fats. Although linoleic acid (an n-6 fatty acid) is perhaps the most abundant polyunsaturated fatty acid in vegetable oils, some oils, including those present in grasses, are richer in α -linolenic acid (an n-3 fatty acid). This is particularly sensitive to peroxidation in tissues and may therefore be more likely to cause problems than linoleic acid (however, α -linolenic acid may otherwise have benefits; see Chapter 5, 'Fat supplements'). The alkali treatment of roughage to improve its digestibility has attracted interest. It must, however, be appreciated that such treatment destroys α -tocopherol and β -carotene, and unless appropriate supplementation is given, signs of myopathy may occur in animals consuming significant quantities of such roughage.

Horses require about 0.15mg available Se/kg feed to meet their dietary requirement for this element, although the breeding mare may need up to 0.25mg/kg dietary DM. Se supplementation is normally given as either sodium selenite, or

sodium selenate, and evidence in horses (Podoll *et al.* 1992) indicates there is no difference between them in potency. Measurement in laboratory animals, however, shows that organic plant sources of Se are more potent than inorganic, although barium selenate and amino acid chelated Se possess a high availability.

Se in forages and seeds occurs as selenocystine, selenocysteine and selenomethionine. Selenium-enriched yeast is more digestible than Se from sodium selenite (Pagan *et al.* 1999b). In a comparison of dietary organic and inorganic sources of Se at 0.15 to 0.6 mg/kg Richardson *et al.* (2003) found that organic sources tended to raise plasma Se concentration, but inorganic sources tended to increase RBC GSH-Px activity. Thus, there seems to be a difference, not only in digestibility, but also in metabolism. An explanation may be that absorption of organic Se is not affected by minerals present in an inorganic form.

Exercise increases the urinary loss of Se from selenite. After absorption red blood cells take up inorganic Se and return it to the plasma during exercise in a reduced form, hydrogen selenide. Some of this, not bound to plasma protein, is excreted through the kidneys and some is transported to the liver to form part of the selenoprotein pool. By comparison, organic Se is transported in the blood bound to amino acids and is less likely to be lost through the kidneys (Pagan *et al.* 1999b).

Janicki *et al.* (2001) found that 3 mg/day organic Se of selenium-yeast, cf. sodium selenite, produced higher plasma, colostrum and milk Se concentrations in breeding mares and higher serum Se concentrations, GSH-Px activity and influenza antibody titre, possibly associated with greater B-cell proliferation, in their foals.

Deficiency

Se deficiency produces pale, weak muscles in foals and a yellowing of the depot fat. It is known that this form of muscular dystrophy in foals is related to a subnormal level of blood Se and a depressed activity of the enzyme GSH-Px. It is essential that pregnant mares receive adequate Se in their diet as their status affects their foals and their performance at parturition, while their milk provides only modest amounts of Se (Lee *et al.* 1995). Deficient pregnant mares ($<0.5 \mu\text{mol Se/l}$ serum), given at least 500 mg vitamin E and 25 mg Se by intramuscular injection two or three weeks prior to parturition, responded with shorter placental retention times and a shorter interval between parturition and effective mating (Ishii *et al.* 2002). Although the foal's serum Se was raised by treatment of their dams it was still deficient.

Serum Se values may fall to $<0.3 \mu\text{mol/l}$ in foals and reduced amounts among TBs in the UK have been associated with poor racing performance (Blackmore *et al.* 1979, 1982). Only in extreme deficiencies, not normally seen in adult animals, is there sufficient muscle damage for extensive membrane leakage of enzymes such as aspartate amino transferase (AAT; EC 2.6.1.1) and creatine kinase (CK or CPK; EC 2.7.3.2) to be detected. The normal peroxidation occurring in muscles after exercise has, however, not been shown to be reduced by supplementary Se. Se deficiency *in vitro* changes neutrophil function and so may depress resistance to infection.

The Se content of herbage, cereals and other crops depends on the Se content of the soil in which they were grown. Areas of deficiency include New Zealand, and low soil concentrations occur in parts of Scotland and the eastern and western states of the USA (Scottish wheat 0.028 cf. Canadian wheat 0.518 $\mu\text{g Se/g DM}$; Barclay & MacPherson 1992). The Great Plains and central southern states of the USA have regions of high Se concentration in crops. These differences are reflected in blood Se concentration, in GSH-Px activity and in performance. In the western USA, concentrations of Se in the blood show a negative correlation with the incidence of reproductive diseases in mares (Basler & Holtan 1981). In this particular study, blood concentrations ranged from 1.2 to 3.1 $\mu\text{mol/l}$ and dietary concentrations ranged from 0.045 to 0.461 mg/kg, i.e. deficient to slightly excessive. Normal blood Se is said to range from 0.8 to 2.8 $\mu\text{mol/l}$ in adult mares (according to Blackmore & Brobst 1981), possibly suggesting that some 'normal' animals may have impaired reproductive capacity owing to Se inadequacy. Amounts of Se in the serum are closely correlated with whole-blood Se, so serum values in the horse seem, on present evidence, to be a good and reproducible measure of status. An increase in dietary Mn from 38 to 50 mg/kg increases both Se retention and blood Se, according to Spais *et al.* (1977).

Toxicity

Se is highly toxic to animals and also to persons handling the salt in highly concentrated forms. The minimum toxic dose of Se through continuous intake is 2–5 mg/kg feed and acute toxicity is caused in sheep given amounts equal to, or greater than, 0.4 mg Se/kg BW in a single dose.

Outside the UK there are areas where soils can contain in excess of 0.5 mg Se/kg and amounts of 5–40 mg/kg DM are found in certain accumulator plants. The predominant forms of Se in these plants are the soluble organic compounds methylselenocysteine and selenocystathionine. Concentrations of up to several thousand mg/kg have been detected in species of milk vetch (*Astragalus*). Various species of woody aster (*Xylorhiza*) and goldenweed (*Oenopsis*), which grow in low-rainfall areas, are also indicator plants, containing relatively high levels of Se. Toxicity is thus more common in dry regions, but horses select grasses rather than these toxic weeds where there is adequate grazing, as the indicator plants are unpalatable. Where grass is sparse, animals suffer from 'alkali disease' in which excessive Se causes a loss of hair on the mane and tail, lameness, bone lesions, including twisted legs in foals, and sloughing of hooves. Plate 3.2 (S. Ricketts personal communication) depicts signs in Se-intoxicated adult horses. The diseased hooves were hollow, indicated by percussion, and there was greying of hair in the mane and tail, a characteristic not previously noted (Plate 3.2a–c). The abnormal hoof horn and hair contained much higher Se concentrations than did the healthy material.

Intoxication involves a steady increase in tissue Se accumulation over months to a plateau at which excretion in urine and faeces keeps pace with intake. Saturation



(a)

Plate 3.2 A selenium-intoxicated horse. (a) Loss and greying of tail hair; (b) crumbly hoof below coronary band; (c) two feet showing healthy hoof wall distal to (prior to) a period of intoxication during which the wall detached from the underlying tissue. (Photographs courtesy of Sidney Ricketts.)



(b)



(c)

values of liver, kidney, hair and hooves are approximately 20–30 mg/kg DM for which Se, replacing sulphur, is incorporated in tissue proteins as selenocystine and selenomethionine. Doses of Se received by intoxicated horses are normally impossible to estimate accurately from field evidence owing to an accompanying loss of appetite. There is no simple remedy for this intoxication apart from removal of the animals from the region if destruction of the seleniferous plants is impractical.

Se and I interactions

Selenium functions not only as a component of glutathione peroxidase (GSH-Px; EC 1.11.1.9) but also as part of the enzyme 1,5'-iodothyronine deiodinase (EC 3.8.1.4), required in the conversion of T_4 to T_3 . As T_3 is the more potent hormone, Se deficiency is said to be associated with human cretinism in northern Zaïre. There do not appear to be accounts of goitre in Se-deficient foals, so that the equine Se requirement for the functioning of this deiodinase may be less than that for normal function of GSH-Px.

The NRC (NRC 1989) dietary recommendations for Cu, Zn, Fe, Mn, Co, I and Se in pregnancy and lactation were supported by the lack of response amongst foals to further supplementation of their dams (Kavazis *et al.* 2002).

Chromium (Cr)

In the 1950s it was shown by Schwartz and Mertz that impaired glucose tolerance in rats could be caused by a deficiency of the glucose tolerance factor, the active constituents of which are trivalent chromium (Cr), niacin, glycine, glutamic acid and cysteine.

Thus, chromium is essential for normal carbohydrate metabolism as a potentiator of insulin action, and so it is found in insulin sensitive tissues where it stimulates glucose clearance. When given as an inorganic supplement, trivalent Cr (III) at dose levels of 300–500 µg Cr/kg diet accelerates glucose clearance, although no improvements occurred in geriatric mares when a supplement of Cr-L-methionine (0.01 mg and 0.02 mg/kg BW daily) was given (Ralston *et al.* 1999). In some species, apparently, Cr improves both cell-mediated and humoral indices of immunity. However, Dimock *et al.* (1999) were unable to demonstrate any such improvement in geriatric mares, whose immunocompetence may have been poor, by supplementation of an alfalfa/maize concentrate with Cr-L-methionine. Trivalent Cr is relatively nontoxic, whereas hexavalent Cr, present in chromates and dichromates and acting as a pro-oxidant, is orally 10–100 times more toxic. Rats show no adverse effect from 100 mg CrIII/kg diet. The recommended safe daily intake of CrIII for humans ranges from 50 to 200 µg.

Organic Cr in the form of the complete glucose tolerance factor, as found in brewer's yeast, may be more potent than inorganic Cr. Barley bread has been traditionally given to individuals suffering from diabetes mellitus in Iraq. It appears that barley is a richer source of Cr than is wheat, samples typically containing over

6mg Cr/kg. Some yeasts are very rich sources of organic Cr, and horses given 5 mg Cr, as yeast, in a natural basal diet providing 12mg Cr/day, showed a decrease in both glucose and insulin responses. During exercise a lower plasma cortisol response and raised plasma TAG concentrations were observed with the high Cr yeast. As insulin inhibits the activity of intracellular hormone-sensitive lipase in adipocytes, the lower insulin response probably allowed more NEFA to be mobilized for both oxidation and return to the liver. There NEFA would be re-esterified, entering the circulation as TAG, contained in very-low-density lipoproteins. Ott & Kivipelto (1999) gave yearling horses coastal Bermuda grass (*Cynodon dactylon*) hay supplemented with a concentrate providing chromium tripicolinate at 0, 105, 210 and 420µgCr/kg total diet. The tolerance to an intravenous glucose injection was improved by the highest chromium supplementation. The Cr would seem to have reduced insulin resistance, reduced stress and facilitated energy metabolism in all these horses. Nevertheless, doubts have been expressed over the safety of picolinate sources in human nutrition.

Nickel (Ni)

Nickel (Ni) is both essential for and toxic to the functioning of ruminal microorganisms, and so the dietary concentration may be critical for the horse. In the human subject toxicity from Ni, CrVI and Fe is mostly confined to pollution from metal industries.

Silicon (Si) and binders

Although Silicon is the second most abundant element in the earth's crust, its absorption may be influenced by dietary Mo and aluminium (Al) and its retention by animals is sufficiently low for it to be classified as a trace element. (NB the best bio-available source of Si in human nutrition is beer!) (Jugdaohsingh *et al.* 2002.) Connective tissue Si is found in osteoblasts. It is a component of animal glycosaminoglycans, and their protein complexes, so it appears to be essential for bone matrix formation. It seems to have a structural function, acting as a cross-linking element in the polysaccharide chains of the proteoglycans linked to the collagen (protein) of cartilage. Collagen synthesis also requires Si for the optimal activity of proline hydroxylase (EC 1.14.11.2). Si is probably also required for bone mineralization, as deficiencies in other species include abnormalities in the skull and leg bones. Independently of vitamin D-induced abnormalities, long bones have a reduced circumference, thinner cortex and reduced flexibility resulting from Si deficiency.

Lang *et al.* (2001) supplemented brood mares at foaling with 220g/day supplemental Si, given as sodium zeolite (hydrated aluminosilicate which breaks down to monosilicic acid and aluminium in the gut). By two months after foaling, increases in Si concentration were found in mare's blood plasma and milk and in the foal's plasma. Si also showed a tendency to increase mare's serum osteocalcin (a

non-collagenous protein synthesised by the osteoblast and used as an indicator of bone formation). The supplementation of growing Quarter Horse foals at 6–18 months of age with sodium zeolite at the rate of 18.6g/kg total diet (Nielsen *et al.* 1993) increased subsequent plasma Si concentration and speed. It also extended work time before leg injury, reducing its frequency and delaying withdrawal from training.

The critical period seems to be up to one year of age in Quarter Horses, as supplementation commencing at the yearling stage of growth had no physiologically important effects on running performance from 18 months of age. Al salts can also protect against toxic metal absorption. Whether this is a factor in the observation of Nielsen *et al.* (1993) is unknown. However, Al and Mo can inhibit tissue accumulation of Si and inhibit bone formation by reducing osteoblastic activity, osteoid mineralization and matrix formation. Whether the Al component plays a useful part by precipitating excess phosphorus in the gut, or whether the toxicity of Al is simply exercised by inhibiting Si use is not known. It should be questioned whether an Al silicate is the most appropriate vehicle for providing Si.

Zeolites are able to exchange constituent cations without a major change of structure. The inclusion of zeolite, in a diet of breeding pigs that also contained zearalenone improved reproductive performance (Papaioannou *et al.* 2002). It is known that zeolite will increase faecal excretion of zearalenone and of aflatoxin B₁.

Frape *et al.* (1981, 1982) demonstrated that a cell wall component of wheat grain (the MAD fibre extract) bound aflatoxin B₁ and greatly increased its faecal excretion. MTB-100 (Alltech, Inc., Nicholasville, Kentucky, 40356, USA), an esterified glucomannan of yeast cell wall, binds aflatoxin, deoxynivalenol, fumonisin, T-2 toxin, ochratoxin and zearalenone (Anon 2002), and clay binders bind aflatoxin. It is possible that a part of the response to zeolites may be explained by their ability to bind intestinal toxins.

Boron, gallium and vanadium

No equine information is available on the elements boron, gallium and vanadium.

STUDY QUESTIONS

- (1) The use and function of calcium is now well understood. What factors affect calcium adequacy and use and why is it associated with a continuing problem in horses?
- (2) Most feeds contain adequate potassium and dietary sources are generally well used. Why is potassium of any concern from nutritional and physiological viewpoints?
- (3) If weanlings were said to have problems with (a) mineral status, or (b) trace element status, how would you set about resolving the position?

FURTHER READING

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Chapter 4

Vitamin and Water Requirements

The drink of all brute creatures being nothing but water, it is therefore the most simple . . . as it is the proper vehicle of all their food, and what dilates the blood and other juices, which without sufficient quantity of liquid, would soon grow thick and viscid.

W. Gibson 1726

VITAMIN REQUIREMENTS

Vitamins are nutrients that horses require in very small quantities, although the actual needs for each differ considerably. For example, the dietary requirement for niacin or for α -tocopherol (vitamin E) may be at least 1000 times the weight of that for either vitamin D or vitamin B₁₂. However, measurements of vitamin requirements lack precision; there is little direct evidence of the requirements for any of the vitamins in the horse and assertions are largely based on measurements in other domestic animals.

Like other mammals, horses require vitamins for normal bodily functions. These requirements will be met by vitamins naturally present in feed, supplementary sources, tissue synthesis and, in the case of vitamin K and the water-soluble B vitamins, additional amounts supplied from microbial synthesis in the intestinal tract. The tissue requirements are complicated by the synthesis of ascorbic acid from simple sugars in the horse's tissues, the production of vitamin D in the skin as a reaction to ultraviolet light, the tissue synthesis of niacin from the amino acid tryptophan, and the partial substitution of a need for choline by methionine and other sources of methyl groups.

Dietary requirements for specific vitamins are therefore affected by circumstance; for example, where horses are kept indoors or are maintained in very high northern latitudes, or indeed have highly pigmented skins and thick coats of hair, their dietary requirements for vitamin D will be greater. Young foals possess a poorly developed large intestine so that little dependence on it for B vitamin or vitamin K synthesis may be assumed. Foals grow fast and, in common with other domestic animals, one must assume that as their tissue requirements exceed those of adults, so their dietary needs are far greater. Tissue demands will also be larger for lactating mares than for barren mares, but as the former are likely to be eating more, this tends to lessen the difference per unit of feed.

Adult horses are able to draw on much larger reserves of some vitamins to see them through periods of deprivation. For example, a good grazing season on high-

quality grass can satisfy the mare's vitamin A requirement through about two months of the ensuing winter. In some instances, however, old mares or other horses have a diminished ability to assimilate nutrients, particularly the fat-soluble vitamins, through a decline in digestive efficiency with age and possibly through the debilitating damage of intestinal parasites. There is also some evidence that the fertility of old barren mares benefits from larger than normal doses of vitamin A. A role for β -carotene, other than as a precursor of vitamin A, is equivocal in the horse, and indeed nothing is directly known of possible functions of the hundreds of other carotenoids in equine metabolism, although some may be suspected.

Inferences drawn from other domestic animals cannot be used in estimating the effect of strenuous work on vitamin needs. It has been asserted, with some justification, that the dietary requirements for certain B vitamins involved in energy metabolism are increased for animals in heavy work, both in total and per unit of feed. This conclusion is reinforced by a frequent decline in appetite during extra hard work. The nutritional requirements of work should, however, not be confused with pharmacological responses. For example, thiamin given parenterally in single doses of 1000–2000 mg is said to have a marked sedative effect on nervous racehorses.

Recommended dietary allowances for vitamins are given in Tables 4.1 and 6.21, p. 227 and a summary of deficiency signs is given in Table 4.2.

Fat-soluble vitamins

Vitamin A (retinol)

Grazing horses derive their vitamin A from the carotenoid pigments present in herbage. The principal one of these is β -carotene and fresh leafy herbage contains the equivalent of 100 000–200 000 iu vitamin A/kg DM for most domestic animals [1 iu equals 0.3 μ g retinol (vitamin A alcohol)]. The horse, however, seems to be

Table 4.1 Adequate concentrations of available vitamins*/kg total diet (assuming 88% DM).

	Mature horse		Mares: last 90 days' gestation	Lactating mare	Weaned foal	Yearling
	Maintenance	Intense work	Stallions			
Vitamin A (iu)	1600	1600	3500	3000	3000	2500
Vitamin D (iu)	500	500	700	600	800	700
Vitamin E (mg)	50	80	60	60	70	60
Thiamin (mg)	3	4	3	4	4	3
Riboflavin (mg)	2.5	3.5	3	3.5	3.5	3
Pyridoxine (mg)	4	6	5	6	6	5
Pantothenic acid (mg)	5	10	5	8	10	5
Biotin (μ g)	200	200	200	200	200	200
Folic acid (mg)	0.5	1.5	1	1	1.5	0.5
Vitamin B ₁₂ (μ g)	0	5	0	0	15	0

*There is no evidence of a dietary requirement for vitamin K, niacin or ascorbic acid in healthy horses.

Table 4.2 Signs of advanced vitamin deficiency in the horse and pony. The status should always be kept well above that leading to these signs to provide positive benefits.

Vitamin A	Anorexia, poor growth, night blindness, keratinization of skin and cornea, increased susceptibility to respiratory infections, infertility, especially in older mares, lameness
Vitamin D	Reduced bone calcification, stiffness and abnormal gait, back pain, swollen joints, reduction in serum calcium and phosphate
Vitamin E	Pale areas of skeletal muscles and myocardium, red cell fragility, reduced phagocytic activity
Vitamin K	Extended blood-clotting time (prothrombin time), but vitamin K deficiency is rarely seen unless it is induced by drugs. Vitamin K is also required for osteocalcin function and bone health.
Thiamin	Anorexia, incoordination, dilated and hypertrophied heart, low blood thiamin and elevated blood pyruvate
Folic acid	Poor growth, lowered blood folate
Biotin	Deterioration in the quality of the hoof horn, expressed as dish-shaped walls that crumble at the lower edges so that shoe nails fail to hold

relatively inefficient in the conversion of β -carotene to vitamin A, and the carotene in good-quality grass or lucerne hay is estimated to possess only a fortieth of the value, weight for weight, of retinol (vitamin A). Although fresh pasture herbage would normally provide well in excess of the requirement, hay used for feeding horses in the UK provides meagre amounts of carotene and particularly where it is more than six months old should be considered to contribute none, unless it is visibly green. Greiwe-Crandell *et al.* (1995) found that reserves in horses deprived of pasture were depleted in about two months.

Various signs of vitamin A deficiency have been recorded, as it has several important functions, among them the integrity of epithelial tissue, normal bone development and night vision. One of the earliest signs of deficiency includes excessive lacrimation (tear production); a protracted deficiency may cause impaired endometrial function in the mare. K.M. Greiwe-Crandell (personal communication) observed increased frequencies of retained placenta at parturition and lower birth weights, slower growth rates and contracted tendons in foals following vitamin A depletion of the dam.

Figure 4.1 indicates that these clinical signs of deficiency occur under fairly extreme conditions of deprivation. As many horses are stabled for most of their time, when they consume little or no fresh herbage, the possibility of this deprived state exists. However, few cases of overt vitamin A deficiency are recognized among stabled horses in Western countries as most routinely receive supplementary synthetic sources. There is evidence of responses in several animal species to rates of intake above the minimum requirement level (Fig. 4.1) under the stress of certain chronic transmissible diseases. Some forms of infertility, particularly in elderly mares, may respond to vitamin A therapy and responses among TBs in training suffering tendon strain and lameness have been noted (Abrams 1979).

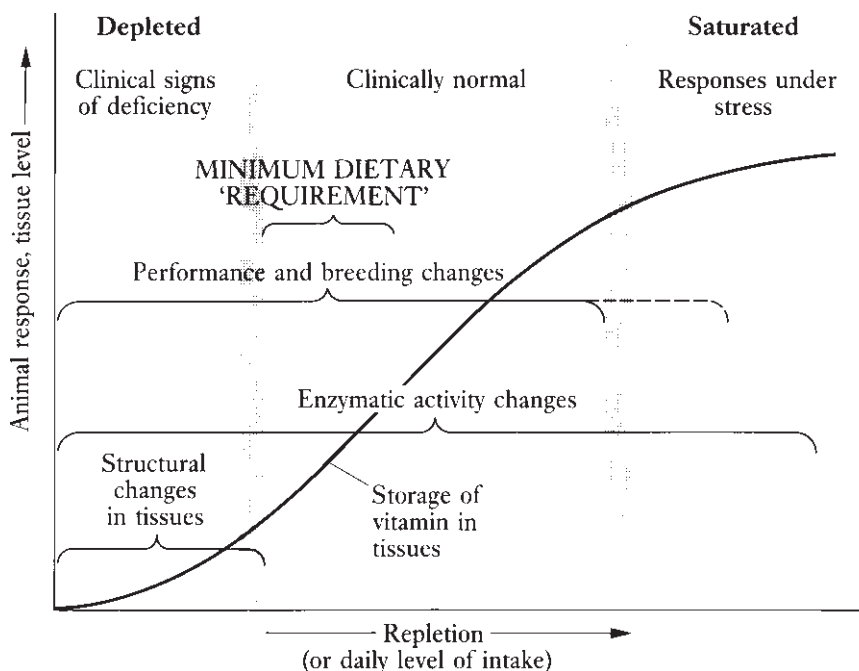


Fig. 4.1 General relationship of animal response to level of vitamin intake.

Measurement of vitamin A status from blood values

Blood plasma retinol has a relatively low concentration in horses. Butler and Blackmore (1982) give a range of 60–300 µg/l for TBs in training in the UK. This range is consistent with that given for other countries, but a marginal dietary deficiency causes little change in this. Plasma retinol is sustained by hepatic reserves and so varies only to a small extent with intake. The vitamin A status can be assessed by use of the relative dose response (RDR). For the horse this is determined in jugular blood as:

$$100(A_4 - A_0)/A_4 = \text{RDR}\%$$

where A_4 is plasma retinol concentration four hours following feeding 10000 iu vitamin A, and A_0 is fasting plasma retinol concentration (Jarrett & Schurg 1987). A deficiency is indicated where RDR% is greater than 20%.

The response at four hours, measured as total vitamin A, probably represents a combination of retinyl ester, in the process of absorption, plus the subsequent release of retinol from the liver. However, the object of the assay is to measure hepatic release of retinol, which peaks at 12–15 hours post-dose of the ester. Thus, a more sensitive RDR test is obtained by measurement of plasma retinol only, by high performance liquid chromatography (HPLC), at 0 hours and at about 14–15 hours following the dose. The revised procedure employs an oral dose of 123.5 mg retinyl palmitate (224152 iu) (Greiwe-Crandell *et al.* 1995):

$$100(A_{14} - A_0)/A_{14} = \text{RDR}\%$$

By this test a deficiency probably occurs when RDR% exceeds 10–12%.

Requirement

The NRC (1989) recommends 9–18 µg/kg BW retinol daily. This is equivalent to 15000 iu daily for a 500 kg horse (or, say, 1500 iu/kg total feed). Most commercial feeds are supplemented with at least 10000 iu/kg, so providing much more on a daily basis, where an allowance of, say, 4–7 kg is given. Donoghue *et al.* (1981), however, reported that the optimum vitamin A intake for normal growth rate of foals was 17 µg/kg BW daily, for adequate liver-secreted serum constituents 65 µg/kg BW daily and for normal haematopoiesis 120 µg/kg BW daily. As 120 µg/kg is equivalent to 15000 iu/kg feed the optimum intake is clearly not well established. Moreover, the presence of respiratory infections may increase the requirement, as these infections are frequently associated with a decrease in plasma vitamin A concentration. A dose rate of 120 µg/kg BW daily is equivalent to 150000–200000 iu daily for a horse, and, at an intake rate five or more times this, toxic reactions should be expected.

Preformed vitamin A and β-carotene, in common with all other fat-soluble vitamins, are unstable, being subject to oxidation, so that natural feed gradually loses its potency. Synthetic forms of vitamin A are stabilized and, when undiluted and stored in reasonable conditions, they retain more than three-quarters of their potency for several years. The grain/protein concentrate portion of the diet supplemented to the extent of 10000 iu vitamin A/kg (3 mg retinol/kg) should allow all foreseeable vitamin A demands to be met (with the possible exception of haematopoiesis in foals, as indicated above). In practice, deficiencies may arise from failure to supplement feed or from the provision of badly stored old feed.

Vitamin A deficiency can also be induced in livestock by other dietary abnormalities. Evidence from several grazing species indicates that signs of deficiency can arise in stock subsisting on poor forage marginal in carotene, which, like much horse hay, contains less than about 7% of crude protein and is deficient in zinc. Authenticated evidence for such interactions in equids is unavailable, but they probably explain the observations of Jeremiah (14:6): ‘The wild asses stand on the bare heights, they pant the air like jackals; their eyes fail because there is no herbage’. Thus, stock under range conditions should also be given adequate supplementary protein and trace elements.

β-Carotene

The plant pigment β-carotene, is the precursor of retinol (vitamin A); however, the pigment appears to function in the animal body independently of this precursor function. There is evidence that cows deprived of β-carotene respond to its supplementation by an improvement in fertility. It may simply function as an intracellular antioxidant, for in rats stressed with high intakes of corn oil the consumption of β-carotene lowered the activity of liver superoxide dismutase. However, the corpus

luteum accumulates β -carotene and 1g/mare daily has been shown to stimulate ovarian activity in both the follicular and corpus luteum phases and to increase circulating progesterone during dioestrus. Treated mares are said to display an enhanced oestrus, an increased pregnancy rate and reduced cycling disorders. This treatment of stabled breeding mares has, in some studies, led to blood β -carotene concentrations similar to those found in pastured mares. However, problem barren mares are said to accumulate lower amounts in the blood. 'Foal heat' diarrhoea of foals is said to be reduced by the elevated blood β -carotene concentrations of their dams.

A report from Edinburgh (Watson *et al.* 1996), on the other hand, indicates that water-soluble synthetic β -carotene is not absorbed by the horse as no increase in plasma β -carotene occurred with dietary supplements of either 1.8 or 10mg/kg BW *per os* daily. Moreover, no change in cyclical ovarian activity, or in plasma progesterone concentration, was produced in that study, and poorer pregnancy and foaling rates, cf. retinyl palmitate, were observed in another study (K.M. Greiwe-Crandell personal communication). Pasture legumes are particularly rich sources of β -carotene and these natural sources are absorbed by horses, although the equine response is relatively low. Mixed grass-clover pasture contains at least 400mg β -carotene/kg DM during the growing season, providing many times the equivalent of the vitamin A requirement. Fonnesbeck & Symons (1967) recorded plasma concentrations of 70 μ g/l β -carotene from daily intakes of 650mg lucerne β -carotene in comparison with 40 μ g/l β -carotene from 170mg β -carotene derived from fescue grass (also see 'Lucerne', Chapter 5).

Breeding mares deprived of pasture during the winter can become depleted of the fat-soluble vitamins A, D and E, as grass hay, or silage, is generally not an adequate source of these vitamins. Blood concentrations tend to be lowest in late winter (Mäenpää *et al.* 1988a); moreover, mares may be depleted of vitamin A two months following removal from pasture, or by mid-winter in pastured mares (Greiwe-Crandell *et al.* 1995).

Vitamins D₂ (ergocalciferol) and D₃ (cholecalciferol)

Function

The widespread occurrence of bony abnormalities in horses and the misunderstandings concerning the interpretation of Ca and phosphate values in blood justify a short summary of the functioning of vitamin D, which has been elegantly unravelled. Vitamin D is required for the maintenance of Ca and phosphate homeostasis, impairment of which produces the lesions in bone called rickets, or osteomalacia and the risks of lameness and bone fractures.

Metabolism

It has been realised in recent years that a fuller understanding of vitamin D function and of the calcium economy of a horse requires an assay of the active form of parathyroid hormone (PTH), secreted by the parathyroid gland in the neck. Stand-

ard assays use an antibody against the carboxy-terminal- and mid-regions, and another against the amino-terminal region of the molecule (human intact PTH), or simply two antibodies against the amino-terminal region (rat assay). Estepa *et al.* (1998) reported that both assays detected a rise in PTH concentration of horse plasma when blood ionised Ca was decreased and a decline in PTH concentration with hypercalcaemia. Modulation of ionised Ca occurs as a consequence of the conversion, under the influence of PTH, of vitamin D to its metabolically active form that has two principal targets in many mammals – the small intestine and bone. The active form 1,25-(OH)₂-vitamin D is derived from both ergo- and cholecalciferol. In this form the vitamin fits the classic model of a steroid hormone, but it is found to be present in extremely low concentrations – 55 pmol/l plasma (approximately 24 ng/l) in horses 1–30 years of age (A. Breidenbach personal communication) (human plasma 20–40 ng/l, 1 ng = 1/1000 µg). The intermediary metabolite 25-OH-vitamin D, formed in the liver, is present in much higher concentrations (in human plasma 15–38 µg/l) and is therefore more easily measured. Horse blood contains relatively low concentrations of the 25-OH metabolite. In Finland, Mäenpää *et al.* (1988b) reported winter mean concentrations of 2.14 µg/l and 8.05 µg/l, respectively, for 25-OH-vitamin D₂ and 25-OH-D₃. In summer, the respective mean values were 2.16 and 16.6 µg/l. The relatively higher 25-OH-D₃ value reflects the synthesis of vitamin D₃ in response to solar ultraviolet irradiation of the skin in the summer, whereas the ergocalciferol vitamin D₂ is a poor source, derived from the action of ultraviolet light on cut leafy forage, i.e. hay.

The low equine concentration of even the 25-OH metabolite in blood, following vitamin D injections (Harmeyer *et al.* 1992), seems to indicate that the hormone has a lesser function in the horse than in other domestic animals, where it is critical for the intestinal absorption of Ca. The 25-OH metabolite is converted to the 1,25-(OH)₂ hormone by 1 α -hydroxylase in the renal cortex, the activity of which was found to be nearly undetectable in the horse (Harmeyer *et al.* 1992). Nevertheless, blood plasma concentrations of Ca in the horse are higher than those in the gut lumen, so that active absorption must be taking place, presumably requiring a Ca-binding protein. In fact vitamin D-responsive Ca-binding protein has been identified in the equine duodenum, but it seems to be of less metabolic significance than in other domestic species investigated. It is possible that dietary vitamin D needs to be given in larger quantities than those recommended, as it is essential in the prevention of dyschondroplasia, as well as ensuring adequate bone ash. Nevertheless, excessive toxic doses cause signs similar to those of a deficiency.

In bone tissue, together with PTH, the 1,25-(OH)₂-vitamin D hormone serves to mobilize bone minerals. In the kidney tubules, PTH stimulates the reabsorption of Ca ions but blocks the reabsorption of phosphate. The vital objective of these two hormones, together with thyrocalcitonin, is to sustain a constant level of blood Ca. It is a fascinating fact that they modulate both Ca and P nutrition, but with different signals. When the diet is deficient in Ca, but adequate in P, then a fall in plasma Ca ions triggers the release of PTH from the parathyroid gland. This stimulates renal Ca reabsorption and the production of the vitamin D hormone. Intestinal absorp-

tion and bone mobilization of both Ca and phosphate are facilitated so that blood Ca and blood pH are returned to normal. Blood phosphate does not rise because of the PTH blocking effect on the renal reabsorption of phosphate.

Thyroid calcitonin counterbalances and modulates the effect of PTH by increasing the net deposition of Ca in bone stimulated by a raised serum Ca. By contrast, deficiency of dietary P depresses blood phosphate, which in turn directly raises ionized Ca in the blood, but also stimulates production of the vitamin D hormone. The combined effect of this is to suppress PTH production, increasing phosphate retention by the kidney (negating phosphate diuresis), stimulating Ca and phosphate absorption from the small intestine. Blood Ca, however, does not rise excessively as the lack of PTH increases the urinary loss of Ca (see also Chapter 11).

It is evident that when vitamin D nutrition is adequate for a given age of horse, but not so Ca or P, blood Ca is held within fairly well-defined limits and phosphate will be more variable. In the absence of vitamin D the efficiency of Ca absorption from the intestinal tract and the mobilization of bone Ca are depressed, so that blood Ca levels will fall. Some mobilization of bone Ca will continue, however, so that osteomalacia, or gradual bone decalcification, occurs in the adult horse and rickets, or reduced calcification of bones, is displayed by the young. On the other hand, some authorities dispute the existence of true rickets in growing horses and vitamin D is probably of less metabolic significance than in the young pig. Nevertheless, a loss of appetite, discomfort on standing, lameness, increased risk of bony fractures and a thinning of the cortex of long bones have been described in foals deprived of sunlight and dietary vitamin D. In young horses, the growth plate (epiphyseal plate) of long bones is irregular, widened and poorly defined and the epiphyses are late in closing.

Dietary requirement

If the cereal/protein concentrate component of the diet is supplemented with 1000 iu vitamin D/kg (25 µg cholecalciferol or ergocalciferol, as 1 iu is equivalent to 0.025 µg cholecalciferol or ergocalciferol), then the daily requirement for vitamin D should be met. Moderately large doses of vitamin D can, to some extent, compensate for low dietary Ca by promoting further Ca absorption, particularly where dietary P is in excess. However, large doses of vitamin D (in excess of 2000–3000 iu/kg BW daily, or in excess of 60000–100000 iu/kg diet) will cause similar signs to those of the deficiency and eventual death, owing to the effect of vitamin D hormone on bone mineral mobilization and a consequential calcification of soft tissue.

Natural sources of toxicity

Several plant species, not found in the UK, actually synthesize this highly active hormone as the calcitropic principal, 1,25-(OH)₂-D₃-glycoside. Thus, horses grazing areas where the plants exist will develop rickets and soft-tissue calcification (for example, *Cestrum diurnum*, a member of the potato family, sometimes incorrectly called wild jasmine, found in Florida and other subtropical states, including Texas and California, causes the condition).

Vitamin E (α -tocopherol)

Vitamin E potency is possessed by several tocopherol and tocotrienol isomers and undoubtedly all these have some antioxidant value in the feed and in the GI tract. However, analysis has shown that α -tocopherol is the only isomer to be found in significant amounts in equine tissues and so is the only form to have significant vitamin potency. Thus, natural forms of vitamin E (D- α -tocopherol and its acetate salt) have a somewhat greater bioavailability in the horse than the racemic mixture of synthetic forms (DL- α -tocopherol and its acetate salt) and free alcohols are superior to the salts (Hargreaves *et al.* 2001). Nevertheless, evidence (Christen *et al.*, 1997) indicates that γ -tocopherol displays an active metabolic role in several species.

As tocopherols have an antioxidant property that protects other substances in food, they are themselves destroyed by oxidation. This is accelerated by poor storage, mould damage and by ensilage of forage or the preservation of cereals in moist conditions. After the crushing of oats or grinding of cereals, the fats are more rapidly oxidized and vitamin E is gradually destroyed unless the material is pelleted. Fresh, green forage and the germ of cereal grains are rich sources of vitamin E, but feeds are frequently supplemented today with the relatively stable acetate ester of α -tocopherol. (See 'Selenium', Chapter 3, for effects of vitamin E/selenium supplements.)

Vitamin E status

Evaluation of the vitamin E status of horses is problematic, owing to a relatively low normal plasma α -tocopherol concentration. The normal range is 1.5–5 mg/l. Adipose tissue of horses contains large quantities (10–60 μ g/g) of α -tocopherol that are not prone to the short-term fluctuations characteristic of blood levels. The wide range indicated for these two tissues is intended to accommodate limited evidence for variation found among breeds. TBs tend to have plasma and adipose tissue concentrations at the lower end of these ranges and overall storage is lower than that for vitamin A.

Vitamin E, ascorbic acid and exercise

Vitamin E adequacy has been measured for many years as the dietary amount required to minimize erythrocyte haemolysis in the presence of dialuric acid, hydrogen peroxide or other haemolytic agents. Early studies (NRC 1978) indicated that the horse required only 10–15 mg/kg diet to ensure this. Limited experimental evidence (Lawrence *et al.* 1978) suggests that vitamin E supplements increase amounts of blood glucose and lactate in exercised horses and may help maintain the normal packed cell volume of the blood. A vitamin E deficiency is known to reduce endurance in rats and the vitamin may be particularly important for extended work. Further evidence indicates that vitamin E supplements result in higher red cell GSH-Px activity following exercise (Ji *et al.* 1990).

Ronéus *et al.* (1986) found that to provide adequate Standardbred tissue saturation with α -tocopherol the daily supplement of DL- α -tocopheryl acetate (all-*rac*- α -

tocopheryl acetate) should be 600–1800mg. This is equivalent to 1.5–4.4mg/kg BW. The question that arises then: Is it necessary to saturate tissues with the vitamin? α -Tocopherol forms an integral part of cellular membranes, where it protects polyunsaturated fatty acids. The peroxidation of these ω -6 fatty acids probably increases during exercise – frequently measured as an increase in thiobarbituric acid reactive substances (TBARs) – yielding n-pentane, a hydrocarbon gas that is excreted in the breath, although an assessment of breath pentane following exercise is complicated by an associated increase in breathing rate. McMeniman & Hintz (1992) recorded an increase in plasma ascorbic acid and a tendency to a decrease in plasma TBARs in exercised ponies supplemented with additional vitamin E. However, the additional amount was only 100iu/day and very few animals were used. TB horses may have higher needs. Schubert (1990) reported that quite large supplementary levels of vitamin E improved the track performance of racehorses. A supplement of 240iu vitamin E/kg feed plus 10g/d ascorbic acid has led to higher blood levels of both α -tocopherol and of ascorbic acid in heavily used polo ponies late in the competition season (Hoffman *et al.* 2001).

Amongst endurance horses, a variable vitamin E intake of 1150–4700mg/day during the weeks prior to an 80km ride resulted in a positive correlation of intake with plasma α -tocopherol concentration, and a negative correlation of intake with plasma CK and AST activities during the ride (Williams *et al.* 2003b). These data indicate that the mean of 5mg vitamin E/kg BW for hard exercise may be inadequate. Moreover, it has been proposed that oxidant/antioxidant imbalance plays a decisive role in the pathogenesis of chronic inflammatory airway diseases. A supplement of vitamin E, ascorbic acid and selenium (Winergy Ventil-ate™, Winergy, Pedigree Masterfoods, Melton Mowbray, Leicestershire, UK) improved exercise tolerance and reduced pulmonary inflammation in heaves-affected horses in remission at rest and after a standard exercise test (Kirschvink *et al.* 2002). Hence, the vitamin E requirement of working horses requires further assessment and may be up to 300mg/kg dietary DM (8–9mg/kg BW daily).

Fat supplementation

There is increasing interest in fat supplementation of diets for exercising horses (see Chapter 9). Many vegetable and fish oils, such as corn, soya or cod liver oil, are rich in polyunsaturated fatty acids and oil supplementation of other species is known to increase the vitamin E requirement. In the horse muscle, TBARs have been shown to rise following this supplementation, despite the naturally high concentration of vitamin E in fresh corn or soya oil and the addition of antioxidants during manufacture of the oils. Clearly, stale, badly stored oils should not be used.

Breeding mares

Vitamin E functions by protecting unsaturated lipids in tissue from oxidation. In conditions where the intake of selenium and vitamin E is low, which can occur on pasture, mares give birth to foals suffering from myodegeneration. Pale areas in the

myocardium and skeletal muscles are apparent on post-mortem examination and muscle-cell damage is seen histologically. If the foals survive, damage is said to be irreversible. Other signs include steatitis, or yellowing of the body fat, and general fat necrosis with multiple small haemorrhages in fatty tissues. As with other causes of muscle damage, the activity of blood CK (EC 2.7.3.2) and AAT (EC 2.6.1.1) rises and probably the fragility of red blood cells increases.

Immune function

Vitamin E is required for normal immune function. Baalsrud & Øvernes (1986) reported that a vitamin E supplement given to oat-fed horses increased their humoral immune response to tetanus toxoid and equine influenza virus. An increase in the phagocytic activity of foal neutrophils has been induced by supplementation. Mares given a traditional mix supplemented with 160 iu vitamin E/kg mix, cf. 80 iu/kg, from four weeks before foaling, tended to have higher serum and colostrum concentrations of immunoglobulins, leading to somewhat higher serum IgG and IgA concentrations in their suckled foals (Hoffman *et al.* 1999). Further work is required, especially in relation to neonatal infections.

Vitamin E in prevention of equine degenerative myeloencephalopathy (EDM) and equine motor neuron disease (EMND)

Two neurological disorders of horses have been recognized to involve α -tocopherol status – equine degenerative myeloencephalopathy (EDM) and equine motor neuron disease (EMND). Oxidative injury of myelinated nerve fibres occurs in both diseases. The sheaths of these fibres are rich in unsaturated fat normally protected by α -tocopherol.

EMND is typified by weight loss, despite normal or increased appetite, and increased recumbency and trembling of proximal limb musculature (Mayhew 1994). The lifting of one thoracic limb from the ground for a few minutes may induce trembling, especially in proximal limb muscles of the opposing thoracic limb. Other signs include hyperaesthesia (hypersensitive response to stimulation) and low head and neck carriage. Affected horses move better than they stand. The clinical signs and neuronal lesions bear some resemblance to the clinical signs and lesions present in autonomic neurons of equine grass sickness (see Chapter 10), apart from the normal feed intake of EMND patients. Muscle atrophy is present in the chronic form of EMND and the tail head is frequently held in a raised position, as neurogenic atrophy from oxidative damage to the somatic ventral motor neuron cells causes contracture of muscle. Abnormal brown pigment deposits may be seen in the retinal fundus, and serum ferritin and hepatic iron are frequently elevated (Divers *et al.* 2001; Verhulst *et al.* 2001). In common with EDM, the disease is typically associated with an absence of access by horses to pasture, with the consumption of poor quality hay and with low concentrations of circulating levels of α -tocopherol. EMND seems to prevail among adult horses of a wide age distribution. Divers *et al.* (1994) recorded EMND in 28 horses, ranging in age from 3 to 18 years, with the highest frequency among four-to-nine-year-olds. Quarter Horses are commonly

affected (Divers *et al.* 2001). Mayhew (1994) considered that EMND may require a neurotoxin to initiate oxidative neuronal degeneration in a pre-existing vitamin E deficient state of the adult horse (also see 'Grass sickness', Chapter 10). Green forage and vitamin E supplementation at 2000iu/day are required when access to green forage/pasture is limited. Standard preparations that include Se would normally lead to selenium toxicosis if the dose provides the required amount of vitamin E.

EDM usually develops in growing horses and is possibly more prevalent in some sire lines. EDM is a diffuse degenerative disease of the spinal cord and caudal medulla oblongata of equids. The disease may arise in foals where breeding horses have no access to pasture, or where large numbers of horses are crowded on poorly productive, often dry, pasture and are given poor sun-baked hay. The clinical signs include an abrupt to insidious, onset of symmetric paresis, ataxia and dysmetria. Stiffness of the limbs of foals and yearlings is seen to persist to adulthood. The signs are present particularly in the pelvic limbs, but also in the thoracic. In the author's experience, EDM has been associated with absence of pasture and the presence of developmental orthopaedic disease (DOD) (see Chapter 8), and signs were not expressed until three to five months of age. Older horses exhibit a striking hyporeflexia over the neck and trunk. Histologically, neuroaxonal dystrophy is widespread, and neuronal atrophy, axonal swellings (spheroids), astrogliosis and lipofuscinlike pigment accumulation are prominent in older horses. The signs should not be confused with those of infections, such as that of West Nile virus infection (Cantile *et al.* 2000). The signs of this include ataxia, weakness and paresis of the hind limbs, occasionally progressing to tetraplegia and recumbency. In EDM, plasma α -tocopherol concentrations range from 1–1.5 mg/l. The condition is vitamin E responsive, where it has not progressed too far, and then plasma levels may rise to 2 mg/l. Selenium does not seem to be involved.

For breeding horses without access to pasture, the daily vitamin E supplementation needs to be high – 2000iu daily for breeding mares. For those presenting signs of ataxia, 6000iu DL- α -tocopheryl acetate/horse daily should be mixed in 30ml vegetable oil (see below) which should be added to 1 kg freshly ground cereal on a daily basis (not stored). Little response is likely for three weeks. Once improvement has been achieved the supplementary dose may be slowly reduced to 2000iu daily. Proof of absorption should be sought by α -tocopherol determinations on blood samples.

The lipids of plant leaves are much richer in n-3 polyunsaturated fatty acids than are those of seeds. The author suggests that the cause of EDM may be a combination of deficiencies of natural antioxidants and of n-3 polyunsaturated fatty acids. This assertion would need testing before recommendations could be advanced. Fish oils are rich sources of the higher n-3 fatty acids. As an intermediary step, vegetable oils, such as rapeseed oil, richer in the lower members of the n-3 series than corn oil, might be used as the carrier for the vitamin E; but the dose of oil would have to be much greater than 30ml – say 200ml daily. Natural antioxidants include the carotenoids. The author has found that control of EDM is assisted by providing a source of these carotenoids, such as mould-free carrots, or dehydrated lucerne, and

Table 4.3 Summary of proposed daily treatment for a horse presenting EDM.

Vitamin E	2000–6000 iu
Rapeseed oil ¹	30–200 ml
Clean, mould-free carrots	2 kg
Dried lucerne ²	2 kg
Riboflavin	12 mg/kg concentrate
Coarse mix diluted with molassed chaff	To appetite, given <i>ad libitum</i> to growing weanlings

¹If fish oil is used, the minimum vitamin E dose should be 4000 iu/day.

²A good source of riboflavin, although the additional 12 mg is an insurance.

the riboflavin supplementation of a concentrate mix should be increased to 12 mg/kg (Table 4.3).

It is commented on above that DOD seems to be present in some EDM cases. To this end, as discussed in Chapter 8, the author has found that the *ad libitum* feeding of growing foals and yearlings is helpful. Most published evidence suggests that this aggravates the condition; however, in several studies including Savage *et al.* (1993a, b) meal-fed and *ad libitum*-fed horses have been compared when given the *same concentrated feed* (this comparison was necessary for the needs of good experimental design), whereas the author's experience indicates that the feed for *ad libitum* feeding should be a coarse mix or a small pellet diluted with molassed chaff, so that the amount taken in at each 'sitting' provides a relatively small amount of energy, akin to nibbling.

'Tying-up'

Some benefit to 'tying-up' or myositis cases, seen, on occasion, after one or two days' rest in hard-worked horses, is said to accrue from treatment with selenium–vitamin E injections (see Chapter 11).

Recommended dietary allowance for normally managed horses

Although more vitamin E may be needed when selenium is deficient, both are required nutrients and the amount of vitamin E that should be present in the diet rises in proportion to the level of dietary unsaturated fats (Agricultural Research Council 1981). Typical rations for horses should contain 75–100 iu vitamin E/kg (1 iu is equal to 1 mg DL- α -tocopheryl acetate, i.e. all-*rac*- α -tocopheryl acetate), although the requirement of very young foals may be slightly greater and that of idle adult horses somewhat less than this.

Vitamin K (phyloquinone)

There are two natural sources of vitamin K. Green plants synthesise phyloquinones (phytylmenaquinone) and bacteria synthesise menaquinones. Vitamin K₂,

prenylmenaquinone-n, along with the B vitamins, is synthesized by functioning gut microorganisms in amounts that should normally meet the horse's requirements. However, this source may be inadequate during the first couple of postnatal weeks, or during extended treatment with sulphonamides. Vitamin K functions in the post-translational carboxylation of specific glutamate residues in at least a dozen proteins, including osteocalcin, a major protein of bone matrix and a protein required for bone development. Higher concentrations of the vitamin are required for the function of osteocalcin than are necessary for its role in blood coagulation, although signs of deficiency have not been observed in horses. Carboxylation allows osteocalcin to bind hydroxyapatite in bone formation. The vitamin K status of growing horses increases with age, as indicated by increased binding capacity of serum osteocalcin (but with lower serum osteocalcin concentrations). The binding capacity increases with earlier weaning, indicating higher vitamin K status than with later weaning (Siciliano *et al.* 1999b), probably reflecting an earlier role for the large intestine.

There is some tissue storage of vitamin K, and, in addition to intestinal synthesis, green forage is a rich source of phyloquinone, so supplementation is unnecessary. No correlation between serum osteocalcin, or its binding capacity, with bone density or health was observed in horses given brome grass hay (2.73 g phyloquinone per kg) and a grain-mix (400 mg phyloquinone per kg) (Siciliano *et al.* 2000).

Airway haemorrhages in bleeders are an expression of blood vessel fragility and not one of a failure in the clotting mechanism and so may not be controlled by vitamin K therapy. It is common for racehorses to present evidence of a mild form of haemorrhaging after races, and exercise had no effect on vitamin K status, expressed as hydroxyapatite-binding capacity for serum osteocalcin (Siciliano *et al.* 1999a).

Water-soluble vitamins

Normal intestinal synthesis plus the quantities naturally present in typical horse feeds seem to meet the maintenance requirements for the B vitamins riboflavin, niacin, pantothenic acid and pyridoxine. Should there be a basic change in diet towards root vegetables and certain by-products in the future, then an increase in supplementary needs might result. The needs of lactating mares and weanling foals should be met if good-quality pasture grass is provided. Additional nutrient demands of exercise are discussed in Chapter 9.

Thiamin

Signs of equine thiamin deficiency have been inferred, to a large extent, from studies in other species. However, a deficiency of this vitamin (Carroll *et al.* 1949) was shown to cause loss of appetite and weight, incoordination of the hind legs, a dilated and hypertrophic heart, a decline in blood concentration of thiamin and in the activity of enzymes requiring thiamin as a cofactor. Cymbaluk *et al.* (1978) made

Table 4.4 Normal blood thiamin concentrations ($\mu\text{g/l}$) in Standardbred horses (Loew & Bettany 1973).

Stallions	2.25 ± 0.16
Geldings	3.03 ± 0.13
Mares and fillies	3.36 ± 0.11
Less than 1 year	2.42 ± 0.26
1–4 years	2.81 ± 0.10
5–10 years	3.53 ± 0.14
10–20 years	3.35 ± 0.17

four Standardbred horses thiamin-deficient by the daily oral administration of 400–800 mg amprolium/kg BW (amprolium is a structural analogue of thiamin). After one to two months, thiamin deficiency signs of bradycardia, ataxia, muscular fasciculations and periodic hypothermia of extremities, blindness, diarrhoea and body weight loss were observed, and erythrocyte transketolase activity was depressed.

Table 4.4 gives normal values for blood thiamin levels. This vitamin has been used in the treatment of ‘tying-up’, but there is no corroboration that a deficiency of it is a cause. Grazing animals on heathland infested with bracken fern (*Pteridium aquilinum*) can exhibit signs of thiamin deficiency if they take to eating bracken. Treatment with thiamin is usually effective. About 25% of the free thiamin synthesized in the caecum is absorbed into the blood and a total dietary level of 3 mg/kg seems to meet the dietary requirement. Whether or not the requirement per kilogram of feed rises during periods of hard work has yet to be demonstrated.

Vitamin B₁₂ (cyanocobalamin)

The cyanocobalamin molecule contains the element cobalt (Co). Cattle and sheep grazing areas deficient in this element develop vitamin B₁₂ deficiency, as the rumen microorganisms are then unable to manufacture the vitamin. Co therapy rectifies the situation. Horses seem to be more resistant to Co deficiency, but undoubtedly they require Co at a minimum level of about 0.1 mg/kg diet for intestinal synthesis of the vitamin, as it is assumed, then, to be absorbed in adequate quantities. Synthesis in foals may be inadequate and, in fact, supplemental vitamin B₁₂ has been shown to increase the blood concentration of the vitamin. It is required for cell replication; thus, a deficiency may cause anaemia and a reduction in the number of red blood cells. Macrocytic anaemia is common to both B₁₂ and folic acid deficiencies through a limitation to DNA synthesis.

Although an overt deficiency has not been produced in adult horses, it has been suggested that a response, including a stimulation to appetite, can be obtained in some anaemic animals. Adult horses in training on high-grain rations may need dietary supplementation because a decline in appetite shown by such horses may

reflect a buildup of blood propionate. This VFA is produced proportionately and absolutely in much greater quantities when diets of this type are consumed, and its metabolism to succinate requires methylmalonyl-CoA mutase (EC 5.4.99.2) that has adenosylcobalamin as a coenzyme. In fact, vitamin B₁₂ deficiency causes an elevation in urine of both methylmalonic acid and homocysteine which distinguishes it from folic-acid deficiency, where this does not occur. Early-weaned foals should receive a supplement of 10 µg vitamin B₁₂/kg dietary DM.

Folic acid (pteroylglutamic acid)

Folic acid is a one-carbon donor in nucleic acid and protein synthesis. Natural folates exist as conjugates of *p*-aminobenzoic acid (PABA) with mono- or polyglutamic acid. Both the stability and the availability to the horse of these compounds undoubtedly vary, but there is a scarcity of evidence relating to equine nutrition. The synthesis of folic acid by gut bacteria from PABA is blocked by sulphanilamide, which has a structure similar to PABA and which combines with the bacterial enzyme required for folic acid synthesis. Thus, enzyme action is inhibited by a substance that combines with it to prevent the formation of a normal enzyme–substrate complex. A Quarter Horse with suspected equine protozoal myeloencephalitis (EPM) was treated orally for nine months with sulfadiazine, pyrimethamine and 19 mg folic acid daily (Piercy *et al.* 2002). She presented with dysphagia, glossitis, neurological abnormalities, anaemia, leukopenia, hypoplastic bone marrow and hypofolic acidemia. The abnormalities resolved after the oral administrations were discontinued and replaced by intravenous administration of folic acid and oral treatment with diclazuril. The folic acid deficiency, induced by prolonged administration of folic acid inhibitors, was exacerbated by oral administration of oxidised folic acid.

The forms of folic acid vary depending on the addition of one-carbon units, the degree of conjugation by glutamyl groups and the degree to which the molecule is reduced by dihydrofolate reductase (to di- and tetrahydrofolate). Folic acid in plants exists in polyglutamated forms and prior to absorption it must be deconjugated by enzymes located in the brush border. Passive and active absorption occur, but for active absorption folate must be in a *reduced* form, the form usually distinct from that in supplements. The fully oxidised form requires reduction by dihydrofolate reductase prior to its active absorption. Thus, frequently, folinic acid, a fully reduced form, is administered. Intestinal folic acid, when in the oxidised form, decreases absorption of the biologically active methyltetrahydrofolate, because the former's affinity for intestinal folate-binding protein is approximately six times greater than that of the latter. Moreover, the combined effect of oxidised folate and an inhibitor of dihydrofolate reductase (pyrimethamine) further intensifies the inhibition of reduced folate absorption. (See also 'Folic acid', Chapter 12). This conclusion probably explains the observation of Ordakowski *et al.* (2002) that horses treated with anti-folate drugs were refractory to the co-administration of natural and synthetic folates.

Folic acid is closely associated with vitamin B₁₂ in single-carbon metabolism. In some domesticated animals, a deficiency causes macrocytic anaemia, through impairment of methionine synthetase (EC 2.1.1.13) function, and so depresses DNA and RNA synthesis. In folic-acid deficient humans and horses (Ordakowski *et al.* 2002), elevated concentrations of homocysteine (but not of methylmalonic acid) occur in blood and urine. Supplementation of stabled TBs receiving an inadequate diet produces an increase in serum folate concentration from about 4–9 µg/l and where blood haemoglobin is low the level may be raised. Australian work on folic acid in the horse supports the author's observations suggesting that there is an increased utilization of folic acid by horses in hard work. Green forage legumes are rich sources of the vitamin, but its availability in some sources is low. As horses required to partake in intensive exercise tend to receive less green forage it is recommended that they receive a supplementary folic acid source, or green forage in their diet.

Folic acid supplementation (25 mg/day, 5 days/week) had no effect on the folic acid status, or submaximal exercise assessment, of adult horses managed on pasture and given hay and a commercial concentrate (Ordakowski *et al.* 2003). Ordakowski *et al.* (2001), found 6.5 mg folic acid/kg DM in an April pasture, at foaling, declining to 2.2 mg/kg DM in June. Mean plasma concentration in TB mares, given supplements containing 1.6 or 1.9 mg folic acid/kg, was 20.6 ng/ml at foaling declining to 17 ng/ml at three to six months' lactation. The mare's milk contained approximately ten times the level in their serum, i.e. 218 ng/ml at foaling declining to 147–196 ng/ml during three to six months of lactation (foal serum levels varied with their dam's status). A daily supplement of 2–3 mg folic acid for weaned foals and working horses is appropriate.

Biotin

Biotin is the only water-soluble vitamin to have brought about clinically observable responses with normal diets in otherwise healthy horses. This may have occurred *because* the biotin contained in wheat, barley and milo (sorghum) grains and in rice bran is almost completely unavailable for utilization. That contained in oats is only slightly more digestible. However, all the biotin in maize, yeast and soyabean is accessible, together with most of that in grass and clover foliage.

Hoof wall disease is common in horses. Slater & Hood (1997) reported that 28% of the horses in their Texas survey had some type of hoof wall problem, largely with an undetermined predisposing cause. Nevertheless, many cases of weak, misshapen, cracked and crumbly hooves, that tend to separate from the sole, have responded to dietary supplementation with biotin. A greater response, as measured by horn hardness, tensile strength and possibly growth rate, has been evoked with 15 mg/horse daily than with 7.5 mg (Buffa *et al.* 1992) (Plate 4.1a and b). Hoof horn and capsule growth rates were also increased over a period of five months when a high-fibre pony cube, containing 100 µg biotin/kg, was supplemented with 8 mg biotin/kg feed (0.12 mg/kg BW daily) (Reilly *et al.* 1999). These supplementary



(i)



(ii)

Plate 4.1a Off-forehooves of 8-year-old Irish chestnut gelding, before (i) and after (ii) receiving 15 mg synthetic biotin/day orally for 13 months.

amounts are considerably larger than should normally be adequate as a daily maintenance dose.

It is also essential that the hooves are properly shaped and trimmed. Long, unpared hooves exert excessive pressure on the heels and this restricts blood flow



(i)



(ii)

Plate 4.1b A 5-year-old TB gelding hack, before (i) and after (ii) receiving 15 mg synthetic biotin/day orally for 5 months. (Photographs 4.1(a) and (b) by kind permission of Norman Comben, MRCVS; Comben *et al.* 1984.)

and hence impedes adequate nutrition of the foot, leading to poor quality and crumbly, unsatisfactory growth of walls, sole and frog. The hoof wall grows linearly from the coronary band at the rate of 8–10 mm/month, so that 9–12 months are required for the wall of the toe to grow from the proximal to the distal border (Pollitt 1990) (the sole and the frog are replaced every two months) and at least 10–12 months are required before a maintenance dose of about 2–5 mg supplementary biotin/horse daily may be allowed.

Comprehensive evidence indicates that a dose level of 20mg/day may be required by large horses, and this for up to three years, for the maximum benefit to be wrought (Josseck *et al.* 1995; Zenker *et al.* 1995). In Josseck *et al.* (1995) plasma biotin concentration in untreated horses was 350ng/l and in treated horses 1000ng/l. Kempson (1987) reported that biotin did not overcome a second hoof defect of poor attachment of horn keratin squames. Supplementary limestone (7.5g/day) with biotin improved this defect. Nevertheless, it is possible that this additional response resulted from the more extended use of the biotin. Yet many diets are deficient in Ca, a deficiency that should be rectified.

Horn is primarily composed of protein, rich in sulphur-amino acids, and many proprietary supplements contain methionine, or methyl sulphonyl methane, as a source of biologically available sulphur (see 'Dietary vitamin and mineral supplements, Chapter 5), in addition to biotin, which may be an asset where a low-protein diet is given. Kempson (1987) also noted infection of the keratin squames by *Bacteroides nodosus* responsive to metronidazole, although these organisms probably represent secondary invaders.

Riboflavin, niacin, pantothenic acid, pyridoxine and lipoic acid

Each of the vitamins riboflavin, niacin, pantothenic acid, pyridoxine and lipoic acid has metabolic functions in the horse; however, no evidence of a dietary deficiency has been established, owing presumably to adequate biosynthesis, e.g. intestinal synthesis by the gut flora. Animals are capable of lipoic acid biosynthesis. Whether supplementation may be of benefit in special circumstances, e.g. ill-health or extreme exertion, has not been established. Thiamin, niacin, riboflavin and lipoic acid are intimately involved in energy metabolism.

Thiamin, as thiamin pyrophosphate (TPP), niacin, as nicotinamide adenine dinucleotide (NAD⁺), riboflavin, as flavoproteins, and lipoic acid all function in the tricarboxylic acid (TCA) cycle for the aerobic combustion of acetate to CO₂ and water with the production of energy, eventually provided to muscle cells as high-energy phosphate in ATP. Pantothenic acid functions as a carrier of acyl groups in the form of CoA, which makes high-energy thioester bonds with carboxylic acids, the most important of which is acetic acid. Acetic acid is formed from the metabolic catabolism of both fats and carbohydrates. Acetyl groups from these sources have to be in the form of acetyl-CoA to be further metabolized in fat synthesis, or for energy production in the TCA cycle.

In carbohydrate metabolism, acetate is derived from pyruvate. Pyruvate requires TPP, or cocarboxylase, the active form of thiamin, for the cleavage of pyruvate (an α -ketoacid), otherwise the formation of lactic acid from pyruvate will be accelerated. In intense exercise, the build-up of lactic acid is a component of fatigue. It is possible therefore that supplementary pantothenic acid and thiamin may promote the aerobic metabolism of pyruvate to acetyl-CoA, reducing lactate production, although there is no evidence that this would occur in horses given normal feeds. A.L. Parker (personal communication) in Kentucky showed that neither acute

(4.5 g/horse), nor chronic (3.0 g/day, equivalent to 5.7 mg/kg BW daily), oral dosing with large amounts of niacin had any influence on various parameters of exercise tolerance.

Vitamin B₁₅ (pangamic acid)

Pangamic acid (vitamin B₁₅) is a term that has been used indiscriminately to describe several products. It is not a definable entity and no substantive data appear to have ever been presented to support claims of beneficial biological effects.

Vitamin C (ascorbic acid)

Ascorbic acid is synthesized from glucose in equine tissues. Dietary sources are very poorly absorbed, and in fact Löscher *et al.* (1984) concluded that where supplementation may be required after surgery and trauma, an i.v. dose of 10 g ascorbic acid was needed to raise the blood concentration. It has been employed by injection in post-traumatic wound infections, epistaxis, strangles and acute rhinopneumonia. Its acidic nature leads to local irritation following subcutaneous and intramuscular administration and so it is administered by i.v. injection for removal of renal calculi. Snow *et al.* (1987) reported that single oral doses of 20 g had no effect on plasma concentration, whereas daily administration of either 4.5 or 20 g ascorbic acid resulted in an increased plasma level after five to ten days. No benefit has been demonstrated from such administration to healthy horses.

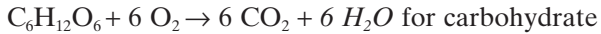
WATER REQUIREMENTS AND FLUID LOSSES

Water constitutes some 65–75% of the body weight of an adult horse and 75–80% of a foal's. Water is vital to the life of the animal. The horse also needs to take in water with its food to act as a fluid medium for digestion and for propulsion of the digesta through the GI tract, for the useful products – milk and growth, and to make good losses through the lungs, skin and in the faeces and urine. In healthy adult horses undertaking light work one estimate showed that the losses of water were distributed such that 18% occurred in the urine, 51% occurred in the faeces and the remaining 31% represented insensible losses (Tasker 1967). Restricted water intake will depress appetite and reduce feed intake.

Equids differ in their ability to conserve body water and to withstand dehydration. Asses from the dry tropics can thwart extreme dehydration because they can conserve water more efficiently than horses. A rise in environmental temperature from 15 to 20°C increases the water requirement of horses by 15–20%. Work, depending on its severity, will raise requirements by 20–300% above the needs for maintenance through increased losses from the lungs and skin. For obvious reasons, peak lactation can lead to requirements twice those of maintenance (Table 7.5, p. 256).

The horse obtains water for its metabolic needs from three sources – the consumption of fresh water, the water content of natural herbage and other foods and from metabolic water. Fresh, young, growing herbage may contain 75–80% water, so that under many circumstances additional fresh water may not be required, but a source should always be provided. In arid conditions herbage is very different, and horses will seek and consume poisonous shrubs and succulent plants unless water and feed are provided.

Metabolic water is that produced during the degradation of carbohydrates, fats and proteins in cellular respiration, e.g.:



where * indicates urea. Thus, per 100g glucose, average fat or average amino acid metabolized, there are, respectively, 60, 106 or 101 g water produced. Per kilogram of feed ingested, it amounts to the equivalent of 350–400 g water, depending on the feed's digestibility. Nevertheless, in circumstances of choice, the water intake of horses is highly correlated with intake of DM and amounts to between 2–4 l/kg DM in stabled horses worked moderately.

Water requirements

Maintenance

For the maintenance of adult horses in an equable environment, the total water requirement is probably less than 2 l/kg DM intake (about 5 l/100 kg BW).

Working horses

Strenuous effort in hot climates increases the water requirement to as much as 5–6 l/kg DM intake (12–15 l/100 kg BW) when there is an inevitable loss of relatively large amounts of sodium and potassium chloride in sweat. Excessive dehydration can be fatal. Certain breeds of horse and other species of *Equus* (e.g. *E. asinus*) (Maloiy 1970) can sustain extensive water loss without apparent discomfort, but horses of temperate breeds may succumb to water losses that amount to 12–15% of their body weight (Hinton 1978; Brobst & Bayly 1982). The estimates of the Hanover group (Meyer 1990) indicate that a 500 kg horse, trotting at 3.5 m/s, in an ambient temperature of 27°C would require a minimum of 10–12 l water/hour to replace inevitable losses. Water repletion should be accompanied by balanced electrolytes, although electrolytes may often have to be given first in order to induce drinking when isotonic or hypotonic dehydration has occurred. Where the horse is fit, it should be walked or allowed to graze so that it cools down gradually over an hour before being given substantial amounts of water. Excessive consumption of cold water by hot horses can precipitate colic or founder. During very cold weather, warm water at a

temperature of 7–18°C should be provided and will be taken more readily than will very cold water. Decreased consumption of water may contribute to the incidence of impaction colic and of depressed performance in racehorses.

The GI tract contains 300 mg Na, 150 mg Cl and 220 mg K per kg BW, or 15–20% of total body Na, 17% of total Cl and 10% of the total K. The GI tract K content is, nevertheless, correlated with intake and the content of all three is related to the GI water content (Meyer 1996). This content is aided by the dietary consumption of soluble fibre, so providing a source of electrolytes and water for extended work (see ‘Endurance riding’, Chapter 9).

Foals and weaned horses

A high-yielding 500 kg mare may produce 12 kg water daily in her milk (see Table 7.5 for water requirements in the stud). However, a foal has a greater requirement than an adult in proportion to its size because it is less able to concentrate urine. A frequent cause of death in neonatal foals is rapid dehydration through persistent diarrhoea, which requires treatment with a physiological salt solution (see Chapter 11). The fluid intake of foals suckling grazing mares was measured (Martin *et al.* 1992) in Queensland, where the maximum ambient temperature averaged 30°C and the relative humidity (RH) averaged 70%. The results are given in Table 4.5.

Weaned horses satisfy their water needs by relatively brief periods of drinking, even when meal-fed. Sufit *et al.* (1985) found that ponies drank for 27 min daily, much of which was periprandial.

Water quality

Where it is feasible, water should be provided from the mains. If mains water is unavailable, then well water, or watercourses, must be free from pollution by sewage or fertilizer seepage. Ideally a new source should be first assessed by a competent analyst. Potentially dangerous microbiological contamination can occur. For instance, the urinary excretion of leptospira by rodents can pollute water, and river and sewage flooding can cause abortion in mares and death of foals. Potomac horse fever is a febrile gastrointestinal disorder of horses caused by *Ehrlichia risticii*. It occurs in North America and Europe between late spring and early autumn in temperate areas and is associated with depression, anorexia, lethargy, fever, mild

Table 4.5 Daily fluid intake per foal (Martin *et al.* 1992).

Age (days)	Milk intake (kg)	Water intake (kg)	Total fluid intake (g/kg BW)	Milk consumed (kg/kg BWgain)
11–18	16.9	nil	246	12.8
30–44	—	3.9	202	15.7
60–74	18.1	5.5	172	16.4

colic, watery diarrhoea, dehydration, oedema and leukopenia. It is maintained in a complex aquatic ecosystem from which transmission to horses occurs by ingestion of insects. Madigan *et al.* (2000) demonstrated its transmission by the ingestion of mature caddis flies from polluted water in northern California.

River water is normally safer than pond water. Nevertheless, in addition to risks incurred from any pollution derived upstream, a decreased rate of flow in summer droughts encourages the growth and accumulation of plant life. Whereas this may assist oxygenation of water, blue-green algae cyanophyceae (Myxophyceae) can proliferate and can be held in position by other plant life. The species that has received most attention is *Anacystis cyanea* (Clarke & Clarke 1975), the consumption of which by horses can cause liver damage, icterus and photosensitization or death. Microcystin, a cyclic decapeptide and an alkaloid hepatotoxin are said to be the toxic agents in this species. There are several other species of algae that contain a variety of toxins, and avoidance of water sources that may contain these species is to be recommended.

Water losses and deprivation

Renal losses

The nonexercised horse disposes of most excess water by excretion through the kidneys. This water is the vehicle for the excretion of excess salts of sodium and potassium, and much of the breakdown products of nitrogen metabolism. Whereas calcium salts may, in part, be excreted in a solid form there is a limit to which the horse can concentrate urea and the highly soluble salts of sodium and potassium. Thus, where diets are rich in salt, or in protein, more dietary water will be required. Based on evidence from other species, an increase in total dietary salt from 7.5 to 30 g/kg would be expected to increase the ratio of free-choice water to DM in the diet from 2:1 to 3.5:1, other things being equal.

Meyer (1990) reported that 73–89% of total water intake was lost by renal excretion in horses given concentrates, whereas <60% was lost by this route in horses given hay. Water restriction decreases renal losses, but does not affect sweat losses in normal horses. Where water restriction persists, then a considerable stress is induced in exercised horses, with an increase in plasma protein and urea concentrations and increased breathing frequency during exercise. Urinary urea at rest, according to the data from Hanover (Meyer 1990), was 6–8 g/l at rest, but the amount increased to 15–50 g/l after exercise, reflecting the solubility of urea and the relative lack of effect on water conservation in some horses. The highest of the urea values occurred with high protein diets and extended water deprivation.

Sweat

Horses lose a large amount of fluid as sweat during exercise. The amount increases greatly with a rise in environmental temperature. Meyer (1990) recorded losses of

Table 4.6 Composition of sweat following 2 hours of exercise (after Meyer 1990).

	g/l		mg/l
Na	2.77	Ca	123
K	1.42	Mg	52
Cl	5.33	Zn	11.4
		Fe	4.3
		Cu	0.27
		Mn	0.16
		Se	2–5 µg/l

Table 4.7 Net water lost per unit volume (m³) of exhaled air and water lost in exhaled air per hour.

	Water lost during expiration (g/m ³)	Expired water lost (g/h/horse)
Resting	38.3	172.5
Trotting 3.5 m/s	44.2	1356.0

11/100kg BW/hour at 18–20°C, but for each degree increase in environmental temperature (range 15–27°C) sweat production increased by 3%. In contrast to fluid losses from the lungs, sweat contains significant amounts of electrolytes and small amounts of trace elements (Table 4.6). During the 2 hours of exercise (Table 4.6) the losses in sweat were calculated to be:

	mg/kg BW
Na	28–69
K	17–30
Cl	56–118

Thus, during this exercise, a 500 kg horse could lose 50–90 g sodium chloride in sweat alone.

Evaporative losses from the lungs

The evaporation of sweat, or of water from the lung surface, absorbs heat and so cools the horse (the isothermal evaporation of 1 kg insensible water absorbs 2256 kJ). The water loss from the surface of the lungs increases greatly during exercise, owing to an increase in body temperature of the horse and an increase in both respiratory frequency and tidal volume. Heat loss through the lungs at 20°C and 60% relative humidity (RH) was calculated to be 289 kJ/hour at rest and 3059 kJ/hour while trotting (Table 4.7).

Table 4.7 indicates that an increase in body temperature during exercise increases the moisture content of exhaled air by 15%, but this, together with the increased respiratory volume during exercise, increases the expiratory water loss nearly eight-fold. Water losses of exercising horses, in sweat and from the lungs, cannot be reduced during water deprivation if body temperature is to be contained within physiological limits, whereas urinary losses are decreased.

Determining water deprivation and response to thirst

Apart from various clinical signs of fluid loss, including skin turgor, the PCV of blood is not a guide to dehydration and water deprivation. The shrinkage of red cells and the changes in the release of red cells from the spleen during dehydration and exercise make this an unreliable guide. Total plasma protein, however, may increase by 10–12 g/l (say, from 62 to 73 g/l) with a fluid loss causing a 12–15% decrease in body weight. Changes in plasma and urine electrolytes and urea depend on several associated factors. In one study (Brobst & Bayly 1982), fluid losses of this extent resulting from dehydration of TB geldings increased the concentration of serum and urinary urea by 68 and 130%, respectively. The specific gravity of urine reached at least 1.042 and urine osmolality increased 30% to 1310 mOsm/kg when the osmolality ratio urine:serum increased to 4.14:1.00. Voluntary drinking has been found to start in ponies when plasma osmolality (mOsm per litre) increased by 3% from normal. Drinking is also stimulated by inducing the formation of urine with a consequential decrease in plasma volume, measured as an increase in plasma protein concentration (Sufit *et al.* 1985).

Nyman *et al.* (2002) compared water intakes of dehydrated, normal and hyperhydrated Standardbred horses following exercise. With both osmotic and hypovolaemic thirst stimulus the two former groups rehydrated more rapidly post exercise than did horses that were hyperhydrated before exercise. The loss of plasma volume was slightly less in hyperhydrated than in normal horses. Plasma aldosterone concentration increased to the same extent after 10 min exercise, irrespective of hydration status; but in one test to a significantly greater extent in the hyperhydrated group. The differences amongst horses in their inclination to drink when dehydrated is likely to have both physiological and psychological explanations. Schott *et al.* (2003) demonstrated three distinct groups amongst 18 untrained two-year-old Arabians in the amount of fluid taken in during and after a 60 km treadmill exercise. 'You can take a horse to water ...'.

The effect of fluid intake before exercise on performance will depend on many factors including the amount consumed, its solute content, length and intensity of exercise and ambient temperature (see also Chapters 9 and 11). Finally it should be noted that horses travelling to a competition may frequently arrive in a dehydrated state.

Table 4.8 Mean fluid contents of the GI tract in ponies given 18 g DM/kg BW as hay or as concentrated feed (after Meyer 1990).

	Hay	Concentrates
<i>3.5 hours postfeeding</i>		
DM (g/kg BW)	19.4	18.3
Water (ml/kg BW)	183	101
Na (mg/kg BW)	398	226
K (mg/kg BW)	289	220
<i>After 1 hour treadmill exercise at 3.5 hours postfeeding</i>		
Reduction in water content (ml/kg BW)	20	8
Reduction in Na content (mg/kg BW)	291	No significant change

Water reserves

Voluntary water intake is greater in horses fed hay than in those given concentrates, and the water content of the GI tract is considerably greater in those given a hay diet than in those given a concentrate mix only. Consequently the GI tract following hay feeding can act to some extent as a reservoir of water and sodium for metabolic needs (Table 4.8).

Sodium chloride dosing is harmful in states of water deprivation, but it will increase voluntary water intake prior to endurance work, increasing water retention. Maximum retention occurred at the 3rd–4th hour after providing salt in a feed (Meyer 1990) and so this may be the optimum time after a small meal for extended exercise during hot weather (see also ‘Long-distance work’, Chapter 9).

STUDY QUESTIONS

- (1) Are there any circumstances in which you might expect a useful response of horses to a fat-soluble vitamin preparation? If there are, describe the circumstances and give reasons why these might exist?
- (2) What factors should be considered in deciding on the adequacy of a water source for horses?

FURTHER READING

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Chapter 5

Ingredients of Horse Feeds

Some parts of the kingdom produce no grain so much as oats which probably may be the reason why they have come to be used as our chief provender.

W. Gibson 1726

Some of the main chemical characteristics of the ingredients of horse feeds are given in Appendix C. Ingredients should be selected not only to provide the nutrients required, but also to be uniform in quality, to avoid harmful contaminants and dust and to balance dense energy-rich feeds with more bulky feeds. The rate of consumption of DE should not be excessive and the stomach contents should retain an 'open' physical texture.

ROUGHAGE

Loose hay

Grasses and forage legumes are cut for hay. Most common species of grass are suitable, but probably the more popular and productive ones include rye grasses (*Lolium*), fescues (*Festuca*), timothy (*Phleum pratense*) and cocksfoot (*Dactylis glomerata*). Many species found in permanent pastures, for example meadow grasses (*Poa*), bromes (*Bromus*), bent grass (*Agrostis*) and foxtails (*Alopecurus*), are also quite satisfactory. Among legumes, red, white, alsike and crimson clovers and trefoils (*Trifolium*), lucerne (*Medicago*) and sometimes sainfoin (*Onobrychis*) are used. Although the crude-fibre content of crimson clover (*Trifolium incarnatum*) hay may be similar to that of other clovers, the fibre tends to be less easily digested by the horse (forage legume fibre generally is more lignified than that of grass). For serving as horse hay or haylage crops, two reliable seed mixtures are:

- (1) three perennial rye grass strains – Melle 5 kg/ha, tetraploid Meltra 15 kg/ha, hybrid tetraploid Augusta 13 kg/ha – or as a two-year crop;
- (2) tetraploid broad red clover – Hungaropoly 7 kg/ha – and tetraploid Italian rye grasses – Wilo 15 kg/ha, Whisper 10 kg/ha.

The leaves of forage legumes and grasses are much richer in nutrients than are the stems, as stems contain about two-thirds of the energy and about half of the protein and other nutrients found in the aerial parts. The leaves of legumes tend to shatter

more readily than grass leaves so that care is necessary at haymaking to conserve the nutritional quality of legume hay. Even so, at the same stage of maturity, legume hay contains more DE, calcium, protein, β -carotene and some of the B vitamins, including folic acid, than does grass hay. Horses consuming hay composed predominantly of forage legume tend to produce more urine with a strong ammonia smell and containing deposits of calcium salts. These events are normal physiological responses in healthy animals.

As long as hay is composed of safe, nontoxic, nutritious plants, the stage of maturity of the crop at the time of cutting and the weather conditions and care to which the haymaking is subject are much more important characteristics than the species of plant present. However, in recent years the digestibility and feeding value of hay made from various grass species have been determined at similar stages of maturity. The digestibility of Matua prairie grass (*Bromus unioloides*) hay, a cool season brome grass, was shown to be higher than that of coastal Bermuda grass (*Cynodon dactylon* L) (Sturgeon *et al.* 1999). Matua brome grass (*B. willdenowii* Kunth) hays, 11.5% and 15.1% CP, were consumed in similar quantities respectively to alfalfa hays of 15.4% and 20.4% CP by breeding mares and the two species led to a similar reproductive performance (Ball *et al.* 1999; Guay *et al.* 2002). As would be expected, Matua grass hay containing 17% CP, 54% NDF, 5.5% lignin has a higher dry matter digestibility than Matua hay of 9% CP, 67% NDF and 9.5% lignin (Box *et al.* 2001). Coastal Bermuda grass (*Cynodon dactylon* L) hay is consumed in greater quantities and produces greater growth rates than hays of either Florakirk or Tifton 85 Bermuda grass, or those of Florona star grass (Lieb & Mislevy 2001).

LaCasha *et al.* (1999) found that voluntary intake and organic matter (OM) digestibility were both greater for the alfalfa (*Medicago sativa* L) hay than for the grass hays: Matua brome grass (*Bromus willdenowii* Kunth. cv. Grasslands Matua) and coastal Bermuda grass (*Cynodon dactylon*), and the intake of the Bermuda grass hay was poorer than that of Matua. The authors concluded that the Matua grass hay should meet the digestible protein and mineral requirements of yearling Quarter Horses. Three other subtropical perennial grass hays: Tifton 85 Bermuda grass, Florakirk Bermuda grass (*Cynodon spp*) and Florona star grass possessed as good, or better, nutrient contents and digestibility cf. coastal Bermuda grass (Lieb & Mislevy 2003).

The voluntary consumption (and digestibility) of warm- and cool-season grass hays as a sole feed varies from 19–29 g/kg BW and is negatively correlated with their NDF content:

$$y = 124.55 + 0.0155x^2 - 2.5742x \quad (R^2 = 0.67) \quad (\text{Lawrence } et al. 2001)$$

$$y = 90.95 - 0.98x \quad (R^2 = 0.68) \quad (\text{Reinowski and Coleman 2003})$$

where x = %NDF in DM and y = g.kg BW⁻¹d⁻¹.

The voluntary intake of three warm-season perennial grass hays decreased in the order: big bluestem (*Andropogon gerardii*), Indiangrass (*Sorghastrum nutans*) and eastern gammagrass (*Tripsacum dactyloides*), (NDF contents 700–740 g/kg DM), but timothy hay (613 g NDF/kg DM) was preferred to all warm-season hays

(Reinowski & Coleman 2003; Reinowski *et al.* 2003). Hay consumption extends eating time and so is beneficial to otherwise idle horses. Those given only timothy hay can spend 6.7–9.3 hours/day eating with a DM intake of 1.5% BW/day (Shingu *et al.* 2001).

As pasture herbage matures, the yield of DM per hectare increases, the moisture content of the crop decreases and, in the UK, the weather becomes warmer. At Jealott's Hill Research Station, Bracknell, Berkshire (ICI Ltd) (owned and operated as a research station by Zeneca CTL, Macclesfield, Cheshire), many years ago, the average yield of DM from early hay crops was only 57% of that cut at a later date. Even when the aftermath was included, the total yield of the early hay amounted to only 71% of that produced by later cutting. Thus, there is a considerable commercial incentive to produce hay composed of grasses at the late-flowering stage. Nevertheless, where hay of good nutritional quality is required for horses, mixtures of grass and clover should be cut before the grass is in full flower when the protein content of the crop lies between 9 and 10% DM and the crop contains high concentrations of calcium, phosphorus and other minerals. Hard, mature grass hays, however, frequently contain 3.5–6% crude protein, lower concentrations of minerals and more crude fibre (Plate 5.1). Hyslop *et al.* (1998a) found that mature pony geldings given *ad libitum* access to mature threshed grass hay (933 g organic matter, 49 g crude protein, 796 g NDF and 6.25 MJ DE/kg DM) were able to exceed by 43% their predicted energy maintenance requirement, whereas the DCP intake was 40% lower than the predicted protein requirement. Many samples of mature grass hay, in my experience, have been similar to this one, requiring a protein supplement for most efficient use, in particular because much of the protein absorbed is inorganic N in the hind-gut.

Using equine faecal flora, Hussein *et al.* (2001) found that dry matter and organic matter digestibilities *in vitro* were lower for perennial rye grass (*L. perenne*), than for brome grass (*Bromus inermis*), orchard grass (*Dactylis glomerata*), or tall fescue (*Festuca arundinacea*) at various stages of maturity. Thus, much further work is required to determine appropriate grass species mixtures for desirable equine pastures and hay, as many features should be assessed.

Good-quality hay from pure stands of lucerne or sainfoin is difficult to make when natural drying is relied on because moisture loss from the thick juicy stems is relatively slow and mechanical turning and tedding can result in a considerable loss of leaf, which dries sooner and shatters more readily. For the best product, these legumes should be cut before flowering at the bud stage, because after first flowering the crude protein content declines at a rate of some 0.5% daily and the digestible energy declines by some 0.75% daily.

Horses should never be given mouldy hay, so the making of satisfactory leafy hays during inclement weather presents a considerable problem, in the absence of a facility for artificial drying. Best-quality hay should be leafy and green, but free from mould dust, weeds and pockets of excess weathering. When ley mixtures are grown for hay, the first cut may contain more weeds, the second cut is generally produced from a faster growth and contains more stem, but the third cut may have the highest nutrient content and leaf, giving a small yield per hectare.

Plate 5.1 Hay samples of various types. (a) Hard 'seeds' hay cut when the grass has formed seed heads. The material is clean, low in dust but of low nutritional value.



(b) Lucerne (alfalfa) hay, which is similarly stemmy and has been sun dried; bleaching destroys its vitamin A potency but it adds some vitamin D potency. Poor harvesting has led to the loss of most of the leaf, so depriving the hay of its most digestible component.



Haylage

(See also Chapter 10, particularly for grass species and safety aspects.) Haylage (grass cut between early silage and hay stages and normally containing, after preservation, 40–65% DM) is being used increasingly, in place of hay, for feeding horses. However, the production of haylage for home consumption can be justified only if:

Plate 5.1 (cont.) (c)

Good-quality lucerne hay, which may have been barn-dried. This is 'rich' material and care should be exercised to avoid loss of leaf during feeding (note the leaf particles at the base of the sample). Artificial drying deprives the hay of vitamin D potency.



(d) Meadow hay containing a proportion of timothy (*Phleum pratense*). This sample is of reasonable quality and is free from significant moulding.



- there is a sufficient number of horses available to make use of the minimum quantity that could be produced in an efficient manner, and a number that could use an opened bale within a couple of days;
- there is adequate grassland, that has not been grazed that year by horses, and that can be set aside and fertilized; and
- there is manpower with the appropriate knowledge, equipment and space available for making, checking and storing the product.

The most successful producers and users of haylage seem to find that a product very high in DM content is the most successful, and acceptable to more of the horses in a stable. At the Yorkshire Riding Centre, in England, meadow grass is cut from mid-June, when the grass is commencing to flower, about two weeks before a hay crop would be taken. The cut material is allowed to wilt in the field for a day and is then square-baled (Plate 5.2) a day before it would be ready as hay, yielding a product with 45–68% DM and 90–120 g crude protein/kg DM. Square bales are preferred as ‘round’ bales are more inclined to mould in the centre, where there may be an empty space. It is important to avoid puncturing the plastic bale cover, as moulding will occur at that point. Yet it is normally safe to pull away and discard only the portion that is mouldy, as the mycelium of *most* moulds is benign and does not penetrate well-compressed bales (Plate 5.2). If there is secondary fermentation, occasioned by excessive heating in the centre, then the whole bale should be discarded. A well-made and packed bale may be left open for up to four days during cool weather, although for a lesser time in hot weather, before the residue should be discarded.

By weight, at 50–60% DM content, haylage is equivalent in energy value to the same weight of typical grass hay. At the Yorkshire Riding Centre 0.405 ha (1 acre) provides a year’s supply of haylage for one horse. About 375 bales meet the requirements of 40 horses for a year, except for one or two that will not eat haylage. The droppings of most horses are looser when they are introduced to haylage, an effect similar to that which occurs when horses are put out to grass, yet no colic should be present. So horses ‘coming off’ grass adapt more easily to a regime that includes haylage. One bale daily is sufficient for about 35 horses in winter, when the horses receive a ration from it on the floor, two or three times during that day (5.5–7.5 kg for smaller horses and up to 12 kg for larger horses daily). This rationing scheme seems to keep the horses in a mental state that is suitable for novice riders. During the summer, when the horses are generally more active, more concentrates are given so that the haylage allowance is reduced by 20–25% and the horses are run on grass at night (Table 5.1). Moreover, competition horses receive less than other horses, in order to avoid an excessively large ‘belly’.

Table 5.1 Daily rations for horses using grass haylage of 50–60% DM.

	Haylage (kg/day)	Concentrates (kg/day)
Event horses	7–7.5	5–6
Riding horses for novices		
1.68–1.73 m (16.2–17 hands) – summer	7–7.5	1.5–2.5
– winter	11–12	0–1
1.57–1.68 m (15.2–16.2 hands) – summer	5–6	1–1.5
– winter	7–7.5	0–0.5
1.42–1.57 m (14–15.2 hands) – summer	5–6	0.5–1
– winter	6.5–7	0–0.5

(a)



(b)



(c)



Plate 5.2 Haylage. (a) A stack of plastic-wrapped bales: square bales are less inclined to mould in the centre than round bales; (b) surface mould on a bale, adjacent to a puncture; (c) a cut section of the bale, showing that mould penetration of the compressed haylage is slight.

Table 5.2 A comparison of grass hay (H), haylage (HY), big-bale silage (BB) and clamp silage (CS) in mature Welsh-cross pony geldings. Feeds were offered at the rate of 1.65 kg DM/100 kg BW daily (g/kg DM unless otherwise stated) (Moore-Colyer & Longland 2000).

	H	HY	BB	CS
DM (g/kg)	922	676	500	337
Organic matter	946	934	937	916
Crude protein	44	70	111	154
Starch	90	50	40	33
GE (MJ/kg)	17.4	17.5	17.8	18.6
Ca	3.1	5.8	4.7	5.5
P	1.3	2.0	3.7	3.3
Mg	1.4	1.8	2.4	2.6
Total NSP	408	293	405	353
DM intake (g/kg LW/day)	14.7	18.4	17.3	9.17
DE (MJ/kg DM)	5.75	9.09	9.83	11.98
DE intake (MJ/kg LW/day)	0.091	0.167	0.170	0.109
DCP (g/kg DM)	6	34	74	104

Table 5.3 Composition, intakes and apparent digestibility coefficients of hay, big-bale grass silage and big-bale red clover silage offered *ad libitum* to ponies (Hale & Moore-Colyer 2001).

	Hay	Grass silage	Red clover silage
Dry matter (g/kg)	852	371	268
Organic matter (g/kg DM)	919	916	866
Water-soluble carbohydrate (g/kg DM)	103	110	8.9
Crude protein (g/kg)	74	104	193
Dry matter intake (kg/day)	5.5	6.1	7.2
Dry matter digestibility	0.36	0.69	0.74
Crude protein digestibility	0.29	0.68	0.80

The intake and apparent digestibility values for haylage, grass hay, big-bale grass and legume silages and clamp silage have been compared using ponies. The DM intake of clamp silage was the lowest, a response that may have been related to its low pH and the DM intake of hay was lower than that for big-bale silages. The digestibility of hay and haylage was lower than for clamp silage and big-bale silage. All feeds but hay, met the daily DE and digestible crude protein requirements (Moore-Colyer & Longland 2000; Hale & Moore-Colyer 2001; Tables 5.2 and 5.3). Subsequent work by this group (Moore-Colyer *et al.* 2003) showed that big-bale grass silage was readily accepted and was more digestible (DM, OM, CP, ADF and NDF) than grass hay derived from the same source, although it produced faeces with a lower DM content and higher pH (Coenen *et al.* 2003c). The comparable DM digestibilities were 67% and 49%

As much of the feeding value of forages is derived from absorption of nutrients from the large intestine, the amino-acid value of forages is low compared with that

of concentrate feeds. The true protein and lysine values of high quality alfalfa cubes are only 60% of the comparable values for a concentrate mixture (Coleman *et al.* 2001).

‘PROCESSED’ FEEDS

Pelleting aids

Common pelleting aids include molasses, lignosulphonite and clay pellet binders (bentonite, hydrated aluminium silicate, principally montmorillonite). Sodium bentonite is also used in feeds as a ruminal fluid buffer and calcium bentonite has been demonstrated to decrease some of the adverse effects of dietary aflatoxin in pigs. Clays have a large surface area and this characteristic, combined with their buffering capacity, is the reason bentonite is included in products to counter laminitis and gas colics of horses (see Chapter 11). It has been considered that clay may inhibit nutrient absorption, but the author's own studies show it does not interfere with vitamin A absorption from a retinyl palmitate source.

Pellets and wafers

Straw, chemically processed with sodium hydroxide or ammonia, will be discussed later in this chapter (see Sodium-hydroxide-treated straw). Hay is also occasionally processed; it may be ground and pelleted or chopped and wafered. During pelleting, molasses and a binding agent are normally added to achieve a satisfactory product. Despite the additional costs of processing, pellets possess a number of advantages:

- The product is easier to weigh and ration.
- There is less waste during feeding and, particularly with leafy legume or grass material, the sifting out of small particles of leaf and their loss in bedding is avoided. This occurs regularly when leafy long hay is consumed.
- Less storage space is required than for long hay.
- Transport costs are lower.
- Horses particularly prone to respiratory allergies (heaves and broken wind) are less subject to dust irritation when given pelleted hay. Coughing is reduced in normal horses and bleeders are less prone to episodes of epistaxis.
- Older horses with poor teeth are inclined to masticate long hay incompletely, so occasionally precipitating colic through impaction. The introduction of pelleted hay should overcome this risk.
- Hay belly may be reduced.

Pellets do, however, have some disadvantages, the principal ones being:

- Incorrectly pelleted material, or good pellets that are allowed to become wet, may be crumbly and soft so that fines are lost, and wet pellets mould within 18–24 hours.

- It is difficult to assess the quality of pelleted hay visually.
- Horses may choke on pellets of about 12mm (0.5in) diameter. The problem is said to be more common when pellets are fed from the hand, but might be avoided by placing a large spherical rock (too large to be chewed) in the manger, forcing the horse to eat around it. The scale of this problem is probably exaggerated and episodes of apparent choking are normally overcome without intervention.
- Wood chewing and coprophagy (faeces eating) are more prevalent where pelleted hay is given without any long hay. The provision of 0.25–0.5kg long hay/100kg BW daily, or good-quality straw bedding, is normally sufficient to minimize these problems. Their incidence seems to be less, but is not eliminated, when wafered hay containing hay particles 4–5cm (1.5–2in) long is used. There is evidence to suggest a relationship between the caecal environment and the incidence of wood chewing in horses given hay or grain. A diet of all hay induces a higher proportion of acetate in the VFA of the caecal fluid (see Table 1.4, p. 17). It has been thought that a low caecal-fluid pH and high propionate content were critical to the inclination for wood chewing (Willard *et al.* 1977). However, the propionate content is unlikely to account for the habit of crib-biters, as these individuals spend less time eating and resting than do normal horses and their reduced feed intake (P.D. McGreevy personal communication) may account for an extended large-intestinal transit time for the passage of ingesta that is likely to be associated with a normal pH (see 'Gastric erosion', Chapters 6 and 11).
- Although the grinding and pelleting of hay do not affect the digestibility of protein, the digestibility of both DM and crude fibre is decreased slightly, possibly because of a decrease in time taken to consume a given amount of feed. From a practical point of view the effect on digestibility is more than offset by the reduction in wastage.
- Grinding and pelleting can increase hay costs by up to 10%.

Feeding time can be influenced by the conditions and method of processing the hay. Researchers in Hanover (Meyer *et al.* 1975b) recorded that horses took 40 min to consume either 1 kg long hay or 1 kg hard, pressed, wafered hay. They took longer to consume chopped or ground hay but less time to eat soft pressed wafers. Hay of poor quality and high fibre content took longer to eat than better quality hay. Highly digestible chopped maize silage was eaten much more rapidly than hay. Horses of between 450 and 500kg made between 3000 and 3500 chewing movements in consuming 1 kg long roughage, but only between 800 and 1200 such movements in eating 1 kg concentrate. However, ponies of between 200 and 280kg required twice as long to eat hay and a concentrate meal and between three and five times as long to eat whole oats or pellets. They made between 5000 and 8000 chewing movements in consuming 1 kg concentrate. The ingesta of horses given chopped hay, or ground hay, passed more rapidly through the stomach than did that of those given long hay and the former led to more fluid stomach contents.

Several other reports have clearly shown that the digesta of horses given ground hay passes more rapidly through the GI tract (Wolter *et al.* 1974), notwithstanding the evidence that horses normally masticate roughage to particles of less than 1.6mm long before it is swallowed (Meyer *et al.* 1975b). In one experiment, the mean rate of passage of long meadow hay was 37 hours compared with 26 and 31 hours for ground meadow hay, and ground and pelleted meadow hay, respectively. The decrease in fibre digestibility experienced on grinding almost certainly is a function of rate of passage through the GI tract. However, by the same token, the faster the rate of passage becomes the greater is the capacity of the horse for feed; but an extension of the time for each meal of pelleted roughage may improve digestibility of fibre.

Where horses are given a choice of loose hay, wafered hay and pelleted hay, more is consumed of the latter two than of the loose hay. Generally speaking, the horse is a reasonable judge of the quality of loose hay and, among grass hays, well-made rye grass is generally reliable. Horses prefer grain to either chopped or long hay, and if given a chopped hay–grain mixture they are inclined to sort out the grain. Nevertheless, such a mixture frequently affords a useful function of depressing the rate of grain consumption by a greedy feeder. Some evidence suggests that the consumption of concentrate feed before hay, rather than after, causes a more intense mixing of ingesta and less variation in the concentration of VFA in the lumen of the large intestine (Muuss *et al.* 1982). This should be an advantage, but other evidence (Cabrera *et al.* 1992) indicates that the consumption of roughage before concentrates improves amino acid utilization from digested proteins.

For stabled horses, long hay is given on either a clean area of the floor in the corner of the box, in a hay rack or in a net. The latter should be placed sufficiently high to avoid the possibility of a horse entangling its hoof in an empty net. The amount of hay wasted may be greatest where it is placed on the floor, but this procedure leads to less atmospheric dust.

Dried grass nuts

Grass, clover, lucerne (alfalfa) and sainfoin crops are frequently cut when green and leafy, artificially dried, and preferably chopped and pelleted with a moisture content of about 120 g/kg. In the UK the product must have a crude protein content of at least 130 g/kg, on the assumption that the moisture content is 100 g/kg, to be designated 'dried grass'. High protein grass nuts contain approximately 160 g protein/kg. Dehydrated alfalfa manufactured in the USA contains 150–170 g protein/kg (90 g moisture/kg). These products contain little vitamin D₂, but are rich sources of high quality protein, β -carotene, vitamin E and minerals, well suited to horse feeding and of relatively balanced composition. However, where the product is rich in legume forage the Ca:P ratio is frequently too wide, and the protein content too high for it to form the entire diet. It should then be supplemented with a cereal product rich in P.

The artificial drying of green forage yields a product more valuable than hay, as the raw material to be dried is less mature, leaves are not shattered and lost,

moulding is avoided and dustiness is minimal. Hyslop *et al.* (1998b) reported that the crude protein and NDF digestibilities were both higher for the artificially dried material than for hay when a perennial-rye-grass crop was cut for both purposes on the same day. It was assumed that there was lower leaf loss for the dehydrated grass. The only disadvantages are the absence of long fibre, and the contents of β -carotene and α -tocopherol are variable and influenced particularly by length of storage of the product. Thus, as much as half the vitamin A potency (initially it may be equivalent to 30 000–40 000 iu vitamin A/kg for horses) can be lost during the first seven months of storage where facilities are not ideal (see 'Feed storage', this chapter).

FUNCTIONS OF HAY AND USE OF OTHER BULKY FEEDS

Haylage

Fibre and bulk are useful attributes for part of the horse's diet. By diluting more readily fermentable material, fibre suppresses a rapid fall in pH of the large intestinal contents and, by stimulating peristaltic contraction, feed with these characteristics probably aids the expulsion of accumulated bubbles of gas. There are many alternatives to hay as sources of fibre and for horses with sound teeth several are useful where reliable hay cannot be obtained. Best-quality silage and haylage free from moulds can be fed to most horses. Good-quality acidified grass silage with a high content of DM may replace between one-third and all horse feed; but success depends on its composition, freedom from abnormal fermentation, skill of the feeder and on the horse. Compensation should be made for its deficiency, compared with grass, in potency of vitamins A and E. Horses suffering from respiratory allergy should benefit most by changing from hay to silage. Very acid silages should be avoided. Silage with low amounts (less than 25%) of dry matter, and baled or bagged material with a higher content of dry matter but with a pH of around 6, may lead to a greater risk of abnormal clostridial fermentation (see Chapter 10). It may also very occasionally precipitate explosive intestinal fermentation and colic if improperly fed. The reason for this may be that the rate of intake of highly fermentable DM is much greater in this form than it is in the form of long hay.

Waste products

Good-quality spring barley or wheat straw in small quantities acts as a source of fibre for horses with sound teeth but is deficient in most nutrients. The inclusion of various waste products in complete diets has been examined, particularly in France and the USA. These products include dried citrus pulp, which is quite satisfactory (Ott *et al.* 1979b), soya hulls and such unusual materials as sunflower hulls, almond hulls, corrugated-paper boxes and computer paper. Digestibilities for the last two seem to be about 90 and 97% respectively, but are much lower for the sunflower and almond hulls because they are heavily lignified. The characteristics of some other bulky feeds are discussed elsewhere in this chapter. With increasing competition

between domestic animals for feeds, and an expanding world human population, undoubtedly the search will continue for satisfactory and safe means of sustaining a healthy population of domesticated herbivores by the greater use of waste products of human activities.

Alkali treatment of roughage

Treating straw with sodium hydroxide – nutritionally improved straw (NIS) – increases its digestibility to the horse (Mundt 1978) and, with dietary adjustments to its sodium content, the product is an important supplier of dietary fibre. Ammonia-treated straw also has promise (Slagsvold *et al.* 1979), but results in cattle and horses have been mixed. The potential digestibility of poor-quality roughages is difficult to predict by chemical analysis, as the factors that inhibit the complete digestion of plant cell-wall polysaccharides include a difference in structural organization, as well as differences in chemical composition of those structures (see Chapter 2).

Group feeding

Feeding habits and hunger of stabled horses can vary enormously and succulent roughages are sometimes used to stimulate animals with flagging appetites. One French study (Doreau 1978) showed that the intake *ad libitum* of a dry feed by a group of stabled horses varied from 8.1 to 19.2 kg daily and the time spent eating ranged from 6 hours 40 min to 15 hours 50 min. The horses ate several large meals and some small diurnal and nocturnal meals. The night meals represented 30% of the total intake. Several factors may contribute to the fastidiousness of finicky eaters, such as environmental stress and nervousness, unpalatability and monotony of ration, nutritional deficiencies, poor health and teeth, lack of exercise and peck order (hierarchy) among group-fed horses.

Succulents

Many succulent vegetables and fruits (for example, sugarbeet roots, carrots, apples, pears, peaches and plums) are satisfactory as treats for horses. Peaches and plums should be stoned, and hard root vegetables should be sliced into strips, to avoid choking, and then mixed with compounded nuts or grain. Carrots contain over 100 mg carotene/kg and care should be exercised in the quantities used (not more than 0.5 kg fresh material/100 kg BW daily) if other large supplements of vitamin A are being given. A similar attitude should apply to all other treats as they represent an unbalanced feed and in large quantities (more than 20% of the total dry matter intake) can do more harm than good. It should also be realized that succulents, including both root vegetables and fruit, contain 80–90% water, and on a dry matter basis they may therefore be a very expensive source of energy and protein. Only if they are relatively cheap can succulents be justified and bulkiness restricts their role to that of a supplement to normal rations. Of all the main flavour groups

present in feed, the horse is deterred by sour tastes and attracted by sweet flavours in moderation.

COMPOUNDED NUTS

Several ingredients are generally incorporated in a ground form, among them the common cereal grains, oilseed meals, milling, brewing and distilling by-products, dried grass and lucerne, fishmeal, and mineral, trace element and vitamin supplements. Their principal role in this form is to provide a balanced source of all nutrients, but they have to be supplemented with loose hay as a source of long fibre, with water and sometimes with common salt. Nuts rich in nutrients and with high digestibility can be supplied to young foals, high-energy nuts can be given to horses in hard work, and low-energy nuts can be provided for adult horses engaged only in light work. The advantages of nuts thus include standardized diets for particular purposes, constant quality, extended shelf life, freedom from dust, palatability, and uniform physical characteristics and density, all of which facilitate routine feeding.

Compounded nuts, particularly high-energy, nutrient-dense formulations, should be introduced gradually to give the horse and its microbial flora time to adapt to the new regime. A too-rapid introduction of nuts, or for that matter of oats, sometimes leads to slightly loose droppings during the first two to three weeks, 'filled-legs' and even to colic. Complete nuts are also manufactured for feeding to horses in the absence of hay, but, generally, these should be used only for a greater control of dust where horses are subject to respiratory allergies. In the absence of loose hay, wood chewing and some other vices, including coprophagy, may be more prevalent. Nevertheless, *ad libitum* access to a complete pelleted diet, one containing 29% crude fibre (53% NDF), enabled three-year-old pony mares to establish natural feeding patterns with minimal vices. Feed intake exceeded maintenance during the first four weeks, but returned to maintenance levels subsequently (Argo *et al.* 2002).

The preparation of feed in nut form may have the disadvantage that the user is unable to recognize good-quality from poor-quality ingredients. Products from reputable compounders should therefore always be used for feeding horses, but some indication of the chemical nature of the product can be conjectured by reference to declared analyses required by law in the EU and found on a ticket attached to the bag. The Statutory Statement included on the ticket should give the following information for complementary and complete feeding stuffs:

- the name or trade name, the price, country of origin and address of the person responsible for the particulars of the Schedule;
- the net quantity and minimum storage life (or batch number), and the moisture content of compounds if it exceeds 14%;
- whether a permitted antioxidant, colourant or preservative is included;

- the active levels of vitamins A, D or E, if included, and the shortest period over which the activities apply;
- the name of any Cu additive and the total level of Cu (naturally present plus added);
- bentonite and montmorillonite, the name of the additive and (other) binding agents present;
- details of any enzymes or microorganisms added (see 'Probiotics', this chapter);
- information may be included on total levels of other trace elements and the total levels of other vitamins, provitamins and vitamin-like substances, including the minimum period over which the activity applies;
- ingredients in descending order by weight;
- the percentage by weight of crude oil (lipids extractable with light petroleum, 40/60°C boiling point without prior hydrolysis, except in the case of milk products);
- the percentage by weight of protein (the nitrogen content multiplied by 6.25);
- the percentage by weight of crude fibre (principally organic substances remaining insoluble following alkali and acid treatment);
- the percentage by weight of total acid-insoluble ash;
- levels of lysine, methionine, cystine, threonine and tryptophan may be included;
- levels of starch, Ca, Na, P, Mg and K may be included (levels of Ca, if over 4.9%, and P, if over 1.9%, must be included).

Table 5.4 gives recommended declarations and some chemical values for compounded horse feeds.

Table 5.4 Recommended declarations and chemical values for compounded nuts and coarse mixtures (assuming 88% DM).

	Crude oil (%)	Crude fibre (%)	Crude* protein (%)	Total lysine (%)	Digestible energy (MJ/kg)	Total ash (%)
<i>Foals</i>						
1 month before weaning						
to 10 months old	4–4.5	6.5–7.5	17–18	0.9	13	7–9
11–20 months old	3–3.5	8.5	15–16	0.75	11	7–9
<i>Adults</i>						
Strenuous work	3.5–4	8.5	12–13	0.55	12	8–10
Light to moderate work, barren mares and stallions	3	14–15	10–10.5	0.45	9	9–10
Last quarter of pregnancy, lactation and working stallions	3	9–10	13–15	0.65	11	8–10

* Actual protein concentrations are less important than are the total lysine contents. Note: lysine and DE are not normally declared.

Table 5.5 Weights of common cereal grains and soya-bean meal and average DE and UFC (MJ/10l) values per unit volume.

	Weights		DE	UFC
	lb/bushel	kg/10l		
Oats	27–45	3.4–5.6	51.7	3.9
Barley	36–55	4.5–6.9	73.0	5.7
Wheat	50–62	6.2–7.7	98.0	7.5
Milo (sorghum)	51–59	6.4–7.4	89.7	6.4*
Maize	46–60	5.7–7.5	93.7	7.4
Soya-bean meal (solvent extracted, 44%)	47–53	5.9–6.6	83.1	5.7
Wheatbran	17–21	2.1–2.6	25.4	1.8

*NE value of milo estimated.

A range of sizes of compounded nuts has been found suitable for feeding to growing and adult horses. However, the optimum seems to be a diameter of 6–8mm and a length of 12mm. For very young foals being given a milk substitute nut, a diameter of 4–5mm and a length of 6–7mm is probably more suitable. South African work (van der Merwe 1975) indicates that acceptability is not affected measurably by hardness of nut, although most horses dislike nuts that crumble too readily, and those that are excessively hard may occasionally be bolted without mastication. This work revealed that smaller nuts are chewed more slowly and require more time for a given amount to be consumed – a decided advantage.

Where horses are in especially hard work, up to 80% by weight of the total ration can be provided in the form of nuts or grain and supplements, with the remaining 20% composed of hay. Regimens of this nature require considerable skill, four to five feeds per day and regular exercise every day. A much more typical regimen for stabled horses in strenuous work is a ration of 50–60% by weight of nuts or concentrate and 40–50% hay. As the amount of work is reduced, so the proportion of nuts can be decreased, or nuts of lower energy can be used. In stables where horses are given their concentrate measured in terms of the number of bowls per day, recognition should be given to the differences in bulk density and energy density of feeds. For example, a unit volume of barley is about three times the weight of the same unit volume of wheat bran. Furthermore, the common cereals have different amounts of digestible energy per unit weight. The combined effects of energy density and bulkiness imply that, for example, a unit volume of maize contains nearly double the digestible energy of the same volume of oats (Table 5.5 gives appropriate conversion values).

COARSE MIXES

Some horses in strenuous work have poor appetites and are more likely to 'eat up' when given a coarse mixture (sweetfeed) than when given nuts in similarly large amounts. Coarse mixtures have thus gained in popularity, although it is evident that poor appetite for nuts is due in part to similar volumes and therefore larger weights of nuts being offered, when refusals would be expected. Coarse mixes should be complete, to be supplemented only with loose hay, water and sometimes with common salt. They tend to be more expensive to produce than compounded nuts, but have an advantage in being less dense, and normally contain a proportion of cooked, flaked cereals and oil seeds and expeller oil-seed cakes. Argo *et al.* (2002) gave pony mares *ad libitum* access to either pellets or a coarse mix of the same composition. They found that DE intake was greater with the pellets, but that each meal was less frequent and of greater duration, with a lower bite rate and a lower DM intake per minute for the coarse mix. Husbandry advantages may accrue from the extension of eating time that occurred with coarse mix. This may explain the greater glycaemic response produced by a 4mm pelleted concentrate compared with that produced by a sweet feed of similar starch content (Harbour *et al.* 2003). On the other hand, their shelf life is less than that attributed to compounded nuts and their bulkiness demands proportionately more storage space. Storage of these mixtures and of all feeds should be in dry, cool, ventilated conditions where there is little variation in temperature, otherwise moulding can occur.

CEREALS

Whereas water is probably the most critical nutrient for the horse's immediate survival, fatness and lack of exercise are the horse's worst enemies. The adequate control of energy intake is the most difficult aspect of optimum and reliable feeding. Cereals (Plate 5.3) are the principal source of energy in the diet of hard-worked horses and therefore a brief discussion of the characteristics of the common cereal grains and their by-products is appropriate.

Cereal grains contain from 12 MJ to more than 16 MJ DE/kg DM compared with about 9 MJ/kg in average hay. Cereal grains embody three main tissues: the husk and aleurone layer, endosperm and the embryo. The endosperm is a rich store of starch required as a source of energy during the early growth of the plant. Of the nitrogen compounds of cereal grains 85–90% are proteins; these are found in each of the tissue regions but are in higher concentrations in the embryo and aleurone layer. Cereal proteins are not as nutritionally useful as oil-seed and animal proteins because they are relatively deficient in lysine, threonine and methionine. The quality of protein (as distinct from the amount) decreases in the order oats, rice, barley, maize, milo, wheat and millet. Oat protein contains slightly more lysine than does the protein of other cereals.

The oil content of cereal grains varies from about 15 to 50 g/kg, with oats containing slightly more than maize, which in turn contains more than barley or wheat. This

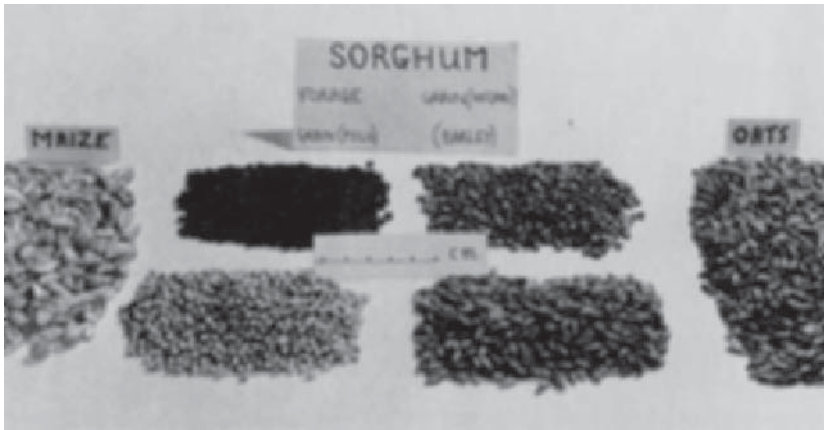


Plate 5.3 Cereal grains. Maize (corn) grains are the largest and may be given whole to horses with sound teeth, but as they can be very hard, cracking is often worthwhile. Barley is smaller and relatively hard and the grains should be crimped or rolled lightly. Oat grains are relatively light and bulky, and crimping or rolling is required only for young horses or for older horses with poor teeth. By comparison, sorghum grains are small, as they are 'naked'. Sorghum is grown in hot dry countries and white varieties are quite satisfactory for horses when coarsely ground, cracked, rolled or cooked. The brown varieties contain large quantities of tannins and are unsuitable for horses.

oil is rich in polyunsaturated fatty acids, of which the principal one is linoleic acid which generally constitutes about half the fatty acid composition by weight in the oil. Unsaturated oils, such as these, are prone to rancidity subsequent to the grinding of cereals unless the meal is compressed into pellets or otherwise stabilized.

Cereal grains are deficient in calcium, because they contain less than 1.5 g/kg, but they have three to five times as much phosphorus, principally in the form of phytate salts. These salts tend to reduce the availability of calcium, zinc and probably magnesium in the intestinal tract; those of oat are said to have a greater immobilizing effect than do the phytates of other common cereals.

Palatability

Although different samples of one cereal species can vary considerably in quality, when given a choice ponies prefer oats to other common cereals. Comparisons made at Cornell (Hawkes *et al.* 1985) showed that the order of preference was oats, cracked maize, barley, rye and cracked wheat. Nevertheless, there was little depression in total intake when only the less palatable cereals were given.

Oats (*Avena sativa*)

Under traditional systems of feeding, in which a single species of cereal grain is given, grains of oat are safer to feed than are the other cereals as their low density and high fibre content make them more difficult to overfeed and the grain size is more appropriate for chewing. They need, therefore, no crimping or rolling for

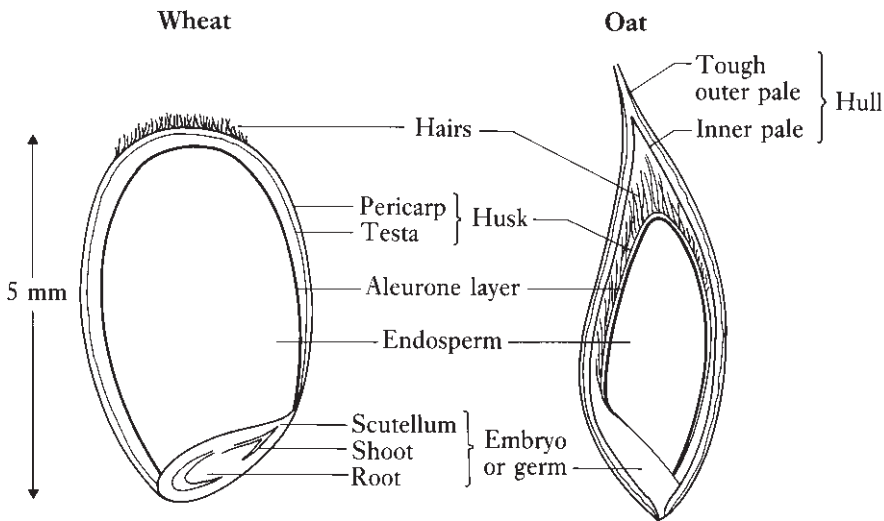


Fig. 5.1 Cross-sectional view through wheat and oat grains.

horses aged over one year if the teeth are sound. A greater quantity of oats than of the other cereals must be consumed to produce founder or other digestive problems. However, they tend to be more expensive per unit of energy than the others, as between 23 and 35% of the grain consists of the hull. Figure 5.1 gives a cross-sectional view of oats and, in Appendix C, their chemical characteristics are listed for comparison with the other common cereals.

Naked oats (varieties produced by crossing *Avena nuda* with *A. sativa*)

The groat of *Avena sativa* is enclosed in a husk that constitutes 250 g/kg of the grain's weight. The husk of the new varieties is loose and falls off during threshing, hence the energy content of naked oats is considerably higher than is that of the grain of *Avena sativa*. Naked oats typically contain 120 g/kg protein and 6 g/kg lysine. The P content is high (3.5 g/kg) but is principally present as phytates and the oil is prone to lipolysis during storage. At present it is advisable to restrict naked oats to 100–200 g/kg of the cereal mix of horses.

Barley (*Hordeum vulgare*)

Oats and barley differ from wheat, maize and grain sorghum in being invested in a hull (botanically known as the inner and outer pales), which the other three cereals have lost during harvesting. All the cereal grains are, however, encased tightly in the thin membrane composed of the less-fibrous fused testa and pericarp (Fig. 5.1).

The barley hull constitutes 10–14% of the total grain weight and the hull is relatively smaller and more tightly apposed to the grain than the hull of the smaller

grain of oats. Thus, for feeding to horses, barley grain should be crimped or lightly rolled to rupture the case shortly before feeding, but it may be fed as the only cereal after a period of gradual adaptation. This period is necessitated by the higher starch content and bushel weight of barley in comparison to oats. Processes that gelatinize starch grains, such as steam rolling or micronization, are discussed later in this chapter (see '(1) Cooking: expansion' and '(2) Cooking: micronization'). Barley protein is of slightly lower nutritional quality than that of oats, being relatively deficient in lysine, and the oil content is quite low, being generally less than 20 g/kg.

Some varieties of naked or hull-less barley, very low in fibre, have been bred. They are comparable with naked oats or oat groats from which the hull has been removed by processing, again yielding a starchy, high-energy, low-fibre product, but their price rarely justifies their use in horse feeding.

Wheat (*Triticum aestivum*)

The grains of wheat (Fig. 5.1) are free of hull and relatively small so that they may escape mastication if fed whole. Wheat should therefore be cracked, coarsely ground or steam flaked before use. The two endosperm proteins (known collectively as gluten) are deficient in lysine and can form a pasty impenetrable mass (similarly for rye grain) for digestive juices, especially when wheat is finely ground. In the uncooked state wheat should form less than half the grain fraction of the diet. Moreover, as the starchy endosperm constitutes 85% of the grain, excessive intakes can cause digestive disturbances, particularly if the adaptation period has been short. The bran and germ make up about 13% and 25% of the grain, respectively.

Triticale

Triticale is a hybrid resulting from crossing wheat (*Triticum*) and rye (*Secale*). It contains more protein than barley, but in variable amounts (100–200 g/kg) which are deficient in the amino acid tryptophan (1.5 g/kg cereal), and richer in lysine than wheat protein. It is said to contain higher concentrations of trypsin inhibitors and alkyl resorcinols than wheat or barley. In ground form it should have a feeding value for horses slightly greater than that of barley. The triticale cultivar Madonna has a higher small intestinal digestibility c.f. wheat grain, leading to a lower caecal starch flow (Brown *et al.* 2001). Some varieties are subject to ergot infection and samples should be examined to ensure there are no significant amounts present.

Maize (corn) (*Zea mays*)

Maize is the largest of the cereal grains and is acceptable in any form for feeding to horses. Frequently, however, when the grains are very hard, they should be cracked, especially for horses with poor teeth. Maize contains twice the energy per unit volume of oats, but oats have a higher small intestinal digestibility, so the glycaemic

index response to whole oats is greater than that to cracked maize (Pagan *et al.* 1999a). A considerable portion of maize starch is fermented in the hind-gut when large amounts are given, unless the grain is cooked (see Chapter 1 and Fig. 6.10, p. 213). The glycaemic response to steam flaked maize is greater than that to either cracked or ground maize (Hoekstra *et al.* 1999), indicating that the cooking improves small intestinal digestibility.

The so-called heating of cereals generally results from the rapid assimilation of their products of digestion and their rapid fermentation by intestinal micro-organisms (with a fall in caecal pH). This causes an abrupt increase in the heat of fermentation and a rise in the concentrations of glucose and VFA in the blood, stimulating metabolic rate to a greater extent than occurs after a meal of hay. Precooking of cereals diminishes the fermentative component of this effect. The grains of maize contain about 650 g starch/kg, but only 80–100 g crude protein. The endosperm protein, zein, is deficient in tryptophan and to a lesser extent in lysine, but the protein of the germ, in common with the germ of other cereals, is of better nutritional quality.

Yellow and white maize varieties are produced, but the types for stock feeding are predominantly yellow and contain the xanthophyll pigments cryptoxanthin, a precursor of vitamin A, and zeaxanthin. Infrequently, some maize imported into the UK is more than two years old, when it is a poor source of vitamin A.

Grain sorghums (*Sorghum vulgare subglabrescens*)

Sorghums are the main food grain of Africa and parts of India and China where they are grown on land too dry for maize. They are also a major stock feed in dry areas of mid-west USA. The kernel is naked, like that of maize and wheat, but more spherical in shape and smaller than that of wheat. The protein content of the grain is variable (80–200 g/kg). The lysine content of the protein is rather low, and the oil content of the grain is less than that of maize. Owing to its size, the grain should always be rolled, cracked, coarsely ground or steam flaked before feeding to horses. This disrupts the waxy bran covering the endosperm. If finely ground it may become pasty and rolling is preferable. It is a high-energy cereal and, therefore, to avoid digestive disturbances, it should preferably form only a portion of the cereal intake.

Several sorghum varieties are grown, including some for forage use. The grains range in colour from white to deep brown. Varieties of grain sorghum that contain only low concentrations of tannins are widely used as a horse feed. Those varieties that are brown, or purple, contain significant amounts of tannins and the white, or milo, grain varieties are the types that should be used (Plate. 5.3). Tannins can cause colic in horses.

Rice (*Oryza sativa*)

The rice grain is invested in a thick fibrous hull, which is easily removed but which constitutes about 20% of the total weight. The hull is rich in silica and when freed

from the grain it is unsuitable on its own for feeding to horses, because the sharp edges may cause irritation. Rough rice, that is the grain before the removal of the hull, is more suitable as a horse feed. Rice protein is a reasonable source of lysine.

Millet (*Setaria* spp., *Panicum miliaceum*)

The name millet is applied to a range of grass species. The seeds have a high energy content, are the palatable staple diet of many people and typically contain 110 g/kg protein of low lysine content, 50–90 g/kg crude fibre and 25–35 g/kg oil. They are free from toxins except for the unripe seed husks of *Paspalum scrobiculatum*. However, millet is not readily available in Europe and parcels that are may have been rejected for human consumption for reasons of mould or admixture. The seed is small and the hull is not removed during normal harvesting. It requires coarse grinding, or crushing, for feeding to horses and its feeding value is somewhat similar to that of oats.

Processing of cereal grains

Specific procedures for processing each species of cereal grain have already been sketched. Where cereals are mechanically rolled the process should be one of kibbling, or bruising, rather than the complete rolling of uncooked material, otherwise the chemical stability of the product is jeopardized, no further increase in digestibility is achieved and greater processing costs are incurred. A similar argument can be advanced for coarse, in contrast to fine, grinding. Moreover, finely ground cereal endosperm is floury, unpalatable, dusty, less stable and may lead to digestive disturbances. Some other feeds such as bran may occasionally ball up and block the oesophagus if fed dry and therefore are normally dampened or mixed with cut or chopped hay, or fed damp with oats. Cereal grains, or high-energy nuts, should be distributed among as many daily feeds as possible to minimize the risk of colic.

(1) Cooking: expansion (extrusion)

Cereals should be cooked only in the presence of water in order to minimize the risk of heat damage to proteins and oil. Steam pelleting and expansion procedures achieve this objective and, for high-energy materials in particular, have been shown to improve the digestibility of dry matter, organic matter, starch and the nitrogen-free extract of cereals and nuts without interfering with the digestibility of crude protein (see Chapter 1). The digestibility of the crude fibre fraction, however, is either not affected or is only slightly depressed if the food material is ground beforehand. This fraction represents only a relatively small portion of high-energy products. The process of expansion, or popping, relies on the cooking effects of superheated steam injected into a slurry compressed against a die face by a revolving worm and the subsequent rapid fall in pressure during extrusion. Material is

subjected to a temperature of around 120°C for about a minute. Processing costs, which constitute more than 10% of the value of the product, are difficult to defend except for young stock and horses under competition rules. It is hard to quantify some of the indirect advantages, which, depending on the process, may include reduced storage space, increased stability of product, improved palatability, destruction of natural toxins, insect pests and bacterial pathogens and the avoidance of high-starch concentrations in the large intestine. The last may be of prime import.

(2) Cooking: micronisation

Other cooking procedures include the traditional steam flaking of maize during which the grain is passed through heated rollers, the roasting of oil seeds during the industrial extraction of oil, and the micronisation of cereals and vegetable protein seeds. For micronization, a moving belt carries a thin, even layer of cereal grain horizontally beneath a series of ceramic burners that emit infrared irradiation in the 2–6µm waveband. This results in a rapid internal heating of the grain and a rise in water-vapour pressure, during which the starch grains swell, fracture and gelatinize. The product is usually then passed through helically cut rollers and from a cooler to a cyclone. The raw material achieves temperatures ranging from 150 to 185°C for 30–70s – for each specific raw material there are optimum values within these ranges. These products are frequently included in coarse mixtures for horses; the process increases digestibility and, for instance, in the case of soya beans, will inactivate antiprotease and other toxic factors.

A high digestibility of starch in the small intestine is important to the avoidance of colic and related metabolic diseases. McLean *et al.* (1998a) incubated monofilament polyester bags containing unprocessed barley, micronised barley and extruded barley in the caecum of ponies offered grass hay *ad libitum*. The initial degradation rate of starch in micronised barley was greater than that of unprocessed barley, or that of extruded barley, and over 40 hours dry matter degradability of extruded material was slightly poorer than for the unprocessed material. The results may indicate a preference for micronisation over extrusion, but increased microbial degradation rate does not necessarily imply better small intestinal digestion and rapid bacterial degradation is likely to promote acidosis.

Subsequently McLean *et al.* (1998b) measured the acetate:propionate ratio of VFA in caecal fluid of ponies given micronised, extruded or rolled barley or hay cubes. The ratio decreased in the order hay cubes > micronised barley > extruded barley > rolled barley. This indicated that greater precaecal starch digestion occurred with micronised barley than with either extruded or rolled barley.

(3) Acidification

The alkali treatment of roughages has been briefly described already (see Alkali treatment of roughage, this chapter). Treatment of high moisture cereal grains with propionic acid has achieved a certain measure of popularity during harvesting in

inclement weather. The acid acts as a mould inhibitor and preservative. Grain treated in this way is only marginally suitable for feeding to horses, owing partly to its acidity and more especially to the frequent presence of mouldy patches in the silo. The grain may become infected by the fungus *Fusarium*, which produces the toxin zearalenone known to cause 'poor doing' in all animals and infertility in breeding animals (see also 'Grass Sickness', Chapter 10). Furthermore, high-moisture cereals are deficient in α -tocopherol so that supplementation with synthetic forms of vitamin E at a level of about 30 mg/kg feed is essential.

Cereals as supplements to hay

C. Drogoul (personal communication) gave ponies chopped meadow hay and rolled barley in the ratios of 100% hay: nil barley, 70:30 and 50:50, at energy maintenance rates, with the barley given before the hay. As the proportion of barley increased, mean retention time increased, but the digestibility of NDF and ADF decreased, probably indicating that $\geq 30\%$ barley led to the presence of starch in the hind-gut and the partial suppression of cellulolytic bacteria (see also Chapter 2).

Cereal by-products

The industrial use of cereal grains leads to the production of two major types of by-product: (1) those derived from the milling industries (the seed coats and germ) and (2) those derived from the brewing and distilling industries (principally spent grains, the residues of germinated grains and dried yeast).

Wheat, oat and rye milling by-products

There are three by-products of oats: the hull, dust consisting of oat hairs lying between the grain and the hull, and meal seeds composed of hull and the endosperm of small seeds. Oat hull has a crude fibre content of 330–360 g/kg with a digestibility little better than that of oat straw. A combination of oat hulls and dust in the approximate ratio of 4:1 gives oatfeed. Each of these by-products may be fed to horses when appropriately processed and included in balanced feeds in proportions of up to 20% of low-energy diets.

Undoubtedly the major milling by-products fed to horses in Western countries are those derived from wheat milling. The offals of wheat consist of the germ, bran, coarse middlings and fine middlings, which comprise about 28% of the total weight of the grain and collectively are known as wheatfeed, although in some products a proportion of the germ is marketed separately. The germ contains 220–320 g crude protein/kg and is a rich source of α -tocopherol and thiamin. This particular by-product is too expensive for general use but can be of value to sick animals. Bran is derived from the pericarp, testa and aleurone layers surrounding the endosperm, with some of the latter attached. It normally contains 85–110 g crude fibre/kg and 140–160 g crude protein. It is sold either as giant, broad or fine bran according to

size, or as entire fraction 'straight-run bran'. These grades are similar in chemical composition, although the larger flaked varieties may contain slightly more water.

Bran is typically expensive for the nutrients it provides, but it can form, as a mash, a palatable vehicle for oral administration of drugs, and it has the capacity to absorb much more than its weight of water. Thus, it has a laxative action on the intestinal tract. Bran in particular, but also other wheat milling by-products, are rich sources of organic phosphorus, as bran contains approximately 10 g/kg, or slightly more, of which 90% is in the form of phytate salts. As bran is deficient in calcium and as phytate depresses the utilization of dietary calcium and zinc, the use of large quantities accelerates the onset of bony abnormalities in young and adult horses.

Coarse middlings are similar to bran but contain somewhat more endosperm and therefore chemically contain only 60–85 g crude fibre/kg and about the same amount of crude protein. Fine middlings contain even more endosperm than the coarse, and consequently only 25–60 g crude fibre. When adjustments are made for the imbalance in minerals, wheat by-products are safe feeds as supplements to horse and pony rations, besides being relatively rich sources of some of the water-soluble vitamins.

Oatfeed

During the commercial preparation of oatmeal for human consumption, the husks, hairs and meal seeds (the husks and part of the endosperm of small grains) are removed. These husks and hairs are combined in a ratio of 4:1 to form oatfeed which in the UK should not, by legal definition, contain more than 270 g crude fibre/kg. Oatfeed is a safe feed used in mixtures to reduce the energy concentration of starch-rich cereals in diets for nonworking horses on the 'easy list'. Oat hulls are rich but variable in lignin content. One study showed this to vary from 9–61 g/kg among worldwide varieties. The raw material is bulky and does not flow easily, so is frequently purchased in a pelleted form by manufacturers.

Rye bran

Rye bran is of limited acceptability to horses.

Maize (corn) by-products

The by-products resulting from the industrial production of glucose and starch derived from maize include the protein gluten, a small amount of bran and the germ. These by-products are similar to the analogous by-products of wheat and frequently all three are combined for sale as maize gluten feed. Although the protein is of poor nutritional quality, the feed is quite suitable as a supplement in horse rations as it is a good source of some of the water-soluble vitamins.

Maize germ meal

The maize germ is removed from the endosperm during the process of corn-starch extraction. Much of the corn oil is then removed from the germ leaving the extracted

maize germ to which may be added other maize offal products, bran and gluten. The composition is therefore variable, depending on the details of the manufacturing procedure. The residual oil is polyunsaturated and thus subject to peroxidation. It is a palatable feed, the protein has a reasonably good amino acid balance and there are no toxic factors present in a well-manufactured and stored product.

Maize gluten feed

Maize gluten is another by-product of the maize starch industry. It is the maize protein, gluten, together with maize bran, extracted maize germ and dried residues of the steeping liquors. The product is rich in xanthophylls, approximately 20 mg/kg, giving it a bright yellow colour. The gluten, free from other by-products, contains over 600 g protein/kg and is available as a branded product, Prairie Meal; but typical material contains 180–230 g protein/kg, 70–80 g crude fibre/kg and only 6–7 g lysine/kg, depending on the amounts of bran and germ remaining. The feed is palatable, free from toxins and a useful constituent of horse feed mixtures.

Rice bran

The quality of rice bran depends on how efficiently the indigestible, siliceous hulls have been removed before the husk is detached from the grain. The bran from the first-stage milling consists principally of the husk (pericarp and testa), the germ and part of the aleurone layer. These components are incompletely removed and the remaining by-product (second-stage milling) constitutes rice polishings, which also contain some endosperm. The first- and second-stage milling by-products are sometimes combined to form rice pollards.

Large quantities of rice meal, or rice bran, are produced globally. This by-product consists of the husk, aleurone layer, germ and some of the endosperm of the rice grain. Inevitably some hull will be present, but this should be at a minimum in the preferred material. The bran has a crude composition of 90–210 g crude fibre/kg, 100–180 g crude protein/kg and 110–170 g lipid material composed of a very unsaturated fat. This fat becomes rancid rapidly and is therefore extracted, leaving a product of much better keeping quality. However, stabilized rice bran is available.

Extracted rice bran is widely available and is a good supplementary feed for horses when used as a component of a mixed ration. The extracted by-product has a composition of about 15 g oil, 130 g crude protein and 120 g crude fibre/kg. However, frequently as much as 60 g silica/kg are present and the ash content of the bran is variable and ranges from 100 to 240 g/kg, reflecting the amount of hull that still remains. Extracted rice bran is also a very rich and variable source of phosphorus (11–22 g/kg), much of which is phytate-P. Care should be taken to ensure that rations in which it is used are appropriately balanced for minerals. Where good-quality rice bran is obtained, 150 g/kg may be included in balanced concentrate feeds. Nevertheless, many Eastern sources send products to the West already seriously contaminated.

Brewing by-products

Three major by-products are derived from brewing: malt culms, brewer's grains and brewer's yeast. When barley is sprouted for the purpose of hydrolysing the starch, the resulting malt sprouts, which include the embryonic radicle (root) and plumule (stem), remain after the malting process. These are removed and dried to form the malt culms. The remainder of the material is mashed to remove sugars, leaving the grains which may be disposed of as a wet by-product or dried and sold as dried brewer's grains.

Malt culms (malt sprouts)

Malt culms (malt sprouts) are the dried shoots and radicle of germinated barley grain. The material contains 12–30 g oil and 140 g crude fibre/kg. The crude protein content is typically 240 g/kg, but is very variable and represented by a proportion of non-protein N. The lysine content is typically 12 g/kg. The by-product is not very palatable, is dusty, and swells on wetting, stimulating peristalsis, but can contain moulds if not fully kiln-dried. An inclusion rate for good material of up to 50 g/kg in horse feeds is satisfactory, but it should not be fed dry on its own.

Dried brewer's grains

The residual grains after removal of the wort may include maize and rice residues in addition to those of barley, the main constituent. The dried by-product contains 180–250 g crude protein and 140–170 g crude fibre/kg and it therefore forms a useful adjunct to mixed horse feeds.

Dried brewer's yeast

The most coveted and expensive by-product of brewing is, of course, yeast, which in dry form contains 420 g high-quality protein/kg and is a rich source of a range of water-soluble vitamins and of phosphorus. This yeast is frequently fed to horses in poor condition at the rate of 30–50 g daily, but is too expensive for regular feeding (also see Probiotics, this chapter).

Distilling by-products

The principal residues from the whisky-distilling industry are the grains and the solubles. The grains in the malt-whisky industry consist solely of barley residues, whereas grain whisky residues may in addition include those of maize, wheat, oats and rice. A proportion of the grains is sold wet, but significant quantities are dried.

Distiller's dried, or light, grains

Distiller's dried, or light, grain is the fibrous residue of barley and of other grains (blended whisky), remaining after washing out the sugars derived from hydrolysis of the starch and used for fermentation to alcohol. For whisky production, the alcohol is distilled off, leaving liquid pot ale from malt whisky production and spent wash

from grain whisky production. This by-product is suitable for inclusion in horse feeds.

Distiller's solubles

After distillation of the alcohol, the spent liquor is spray-dried to yield a light-brown powder known as distiller's solubles, quite suitable for use in mixed feeds. Frequently the dried solubles are added back to the dried grains and marketed as dried distiller's grains with solubles, known also as dark grains.

Distiller's dark grains

The liquid pot ale (or spent wash from maize and other grain distillation) contains unfermentable carbohydrates and products of yeast metabolism, such as protein and vitamins. Where maize is used for alcohol production, this liquid contains maize oil and after evaporation of water and addition of lime the residue is spray-dried. With the grains, the residue produces distiller's dark grains (DDS), or distiller's dried grains with solubles. This by-product in small quantities is a valuable supplement to mixed horse feed and American evidence suggests that dried maize grains with solubles stimulate the digestion of cellulose by microorganisms in the horse caecum.

Malt whisky DDS from barley contains about 270g digestible carbohydrate/kg and 70g ether extract/kg, whereas grain DDS from maize contains about 180g digestible carbohydrate/kg and 110g ether extract/kg. The crude fibre content of DDS is 100–130g/kg, lipid 100–120g/kg, crude protein 260–280g/kg and the lysine content about 8g/kg. The by-product from grain whisky is generally more digestible, but both DDS by-products are low in sodium, potassium and calcium. Both are free from toxic constituents, and rich but variable in copper, malt DDS containing about 40mg/kg, and grain DDS 80mg/kg, and both are suitable for inclusion in horse feeds.

Cereal grain screenings

Cereal grain screenings are residues from the preparation, storage and treatment of cereal grains (barley, wheat, maize, sorghums and soya beans) and their products. They are extremely variable in quality and include broken grains, chaff, weed seeds and dust, and therefore moulds. Mycotoxins may be present. Each batch may differ and their use should be in the pelleted form and strictly limited. The ash content varies from 20–180g/kg. Higher proportions of ash indicate greater soil contamination, and the presence of broken grains contributes to an accelerated rate of rancid cereal oil formation, particularly important in respect of maize.

Chaff and molassed chaff

Chaff is composed of the husk, or glumes, awns and other fibrous waste material derived from the threshing of grain. It is highly lignified, and therefore not well fermented by intestinal microorganisms, but oat chaff, which is the best, is

better utilized than is straw alone. That derived from barley threshing contains large amounts of barley awns, which have serrated edges and may cause some irritation. Nevertheless, it is a safe material to dilute energy-dense cereal grains and is frequently marketed mixed with molasses at a concentration of about 500 g/kg. This product is palatable to horses and overcomes any dustiness of the chaff. Some molassed products also include about 20 g/kg limestone, as a source of calcium; however, it should be borne in mind that these products are deficient in phosphorus. Some chaffs marketed for horses are based on chopped grass hay, 2 cm in length, mixed with molasses and limestone.

Sodium-hydroxide-treated straw

Sodium hydroxide (NaOH)-treated straw is an adjunct to mixed horse feeds and provides a useful source of palatable fibre. It should be recognized that NaOH-treatment of cereal grains, or of other materials that are a source of vitamin E, will destroy the vitamin. Where the feed is also deficient in selenium, nutritional degenerative myopathy can result.

Hydroponics

The practice of germinating barley seeds in lighted trays under humid conditions produces a high-quality feed as rapidly growing young plants. However, the cost per unit of dry matter in particular, owing to the inclusion of a realistic rate of depreciation for capital equipment and the high labour commitment, make the practice difficult to justify on economic grounds. The barley grains should not have been through a grain drier, which would severely damage their capacity for germination, nor should they have been treated with mercurial seed dressings. They should be bright and free from broken grains. The time interval from germination to consumption should be minimized by establishing optimum conditions for growth of 20 hours light per 24 hours and an ambient temperature of 19–20°C. Slow growth increases the likelihood of moulding and its attendant risks. A build-up of mould spores in the room must be avoided by routine hygiene. About three-quarters of the product is moisture and therefore the DE content is only 2.5 MJ/kg despite its high digestibility (Table 5.6). In comparison with pasture grass, hydroponic barley has a very low

Table 5.6 Typical analytical values for hydroponic barley (g/kg DM).

Crude protein	120–170
Crude fibre	80–155
Ash	27–42
Digestible carbohydrate	550–740
Calcium	1.1–1.3
Magnesium	1.5–2
Potassium	4.6–4.8
Phosphorus	4.4–5.5

content of calcium and potassium, whereas the phosphorus contents are similar. The Ca:P ratios are therefore quite different from those of grass.

OTHER LESSER INGREDIENTS AND BY-PRODUCTS

Carob-seed meals (Ceratonia siliqua) and locust beans (Parkia filicoidea)

The evergreen carob tree (*Ceratonia siliqua*) grows in Mediterranean countries. Its green pods fall from the tree in the autumn and are harvested. The hard seeds are embedded in the thick, fleshy maturing pods that are unsuitable for grinding, owing to their high moisture content. They are kibbled, or crimped, and the seeds fall out. These are used in the confectionery industry, leaving the pieces of pod that are distributed to animal feed manufacturers. These dark-coloured pieces are very sweet, containing 400–450 g sucrose, 35 g crude protein, about 70 g crude fibre and 160–240 g moisture/kg. It is not advisable to use batches containing more than 200 g moisture/kg. The pods also contain some tannic acid, but the best sources are a useful feed for inclusion in coarse mixes. Major sources in Europe are Spain, Portugal and Cyprus and the best quality may be derived from the latter source.

The West African locust-bean pods, from a spreading tree (*Parkia filicoidea*), contain dark-brown, sweet, fibrous seeds in fibrous pods that are free from undesirable chemicals. The pod and seed, containing about 300 g sucrose/kg, are drier than those from the Mediterranean area and so can be ground, bulk handled and used in ruminant and horse rations.

Biscuit meal

Bakery wastes are variable and high in energy content, owing to a wide variation in fat content (5–280 g/kg) and high-energy digestibility. In addition to wheat and other flours the wastes contain sugar and salt, which is of no disadvantage to horses. Rancidity and moulding are typical problems and a perennial issue for the Rules of Racing and of Competition is the inclusion of chocolate products in the wastes. In the past, this has frequently been the source of theobromine detected in urine.

Molasses

The crystallization and separation of sucrose from the water extracts of sugar beet (*Beta vulgaris saccharifera*) and sugar cane (*Saccharum officinarum*) leave a thick black liquid, termed molasses, which contains about 750 g DM/kg, of which about 500 g consists of sugars. The crude protein in molasses is almost entirely non-protein nitrogen and of minimal value in feeding horses. In beet molasses, a proportion of this is in the form of the harmless amine betaine (a substitute for choline), which is

responsible for the somewhat unpleasant fishy aroma associated with that form of molasses, but cane molasses has a very pleasant smell. The sweet taste of both forms is attractive to horses when used in mixed feeds up to a level of 100 g/kg feed, and in these proportions molasses can act as a relatively effective binding agent in the manufacture of nuts. Cane molasses contains 5–11 g calcium/kg and the potassium content ranges from 20–40 g/kg in cane molasses and from 55–65 g/kg in beet molasses. Cane molasses is reasonably rich in pantothenic acid and both contain around 16 mg niacin/kg.

Molassed and unmolassed sugar-beet pulp (*Beta vulgaris* var. *saccharifera*)

The shredded residue resulting from the extraction of the sucrose contained in the root of sugar beet is a wet product containing 750–900 g water/kg. This could be ensiled for horse feeding, but it is rarely used in this way. Most beet pulp is dried and marketed as dried sugar-beet pulp, or mixed with molasses to form molassed sugar beet pulp, providing sugar, pectin and betaine, and sold normally in a cubed form. Sugar-beet-pulp shreds containing 10% molasses and whole oats yield similar glycaemic responses. The response is less, but considerable, with soaked pulp or expanded sugar beet pulp (Coenen *et al.* 2003b), in the absence of the molasses, owing to the presence of residual sugar in the pulp. The areas under the glucose response curve as a percent of that to oats were 92 for molassed pulp and 72 for unmolassed soaked pulp (Groff *et al.* 2001).

Hyslop *et al.* (1998c) gave mature Shetland ponies 2 kg DM/day as threshed grass hay and *ad libitum* access to either soaked molassed or unmolassed sugar-beet pulp. The total dry matter intake was lower than for ponies offered threshed hay only, although the DM intake provided 15% more DE than was required for maintenance. More unmolassed pulp was consumed than molassed, and the unmolassed material yielded higher values for DM, CP and NDF digestibilities. Moreover, the partial replacement of oats by unmolassed sugar-beet pulp, at concentrations of 152 g and 108 g/kg DM, has little effect on energy digestibility (Lindberg & Karlsson 2001), although the energy sources differ metabolically. Sugar-beet pulp has a high apparent digestibility and appears to limit total feed intake, but molasses would seem to reduce the digestibility of fibre.

The dry cubes should not be given to horses on their own, without soaking, as in the dry form they can cause choke in some animals (see Chapter 11) and if dried beet pulp constitutes a high proportion of the concentrate mix then large quantities of free fluid are absorbed by the mass in the stomach and colic may result. The carbohydrate it contains is rich in pectins, but also cellulose and hemicellulose that are fermented by the large intestinal flora of the horse. The residual sucrose present is digested in the small intestine. Mixed with other feeds, the dried form is used successfully in coarse mixtures and larger quantities, soaked, act as a useful cereal replacer. It has a uniform composition, is free from toxins and much less likely to cause laminitis than equal quantities of cereals (see 'Laminitis control', Chapter 2).

It contains more calcium than do cereals, on average 6 g calcium/kg, and <1 g phosphorus/kg, but it is a poor source of some vitamins, cf. cereals. The protein content and quality are similar to those of cereals. Urea is sometimes added to beet pulp. This is of no value to horses, but causes no harm in those with fully functioning kidneys. There are no undesirable natural chemicals in beet pulp.

Dried lucerne (dehydrated alfalfa)

Although not a by-product but a useful forage, dried lucerne is mentioned here as several indirect effects have been attributed to it, possibly caused by unidentified factors. Its meal stimulates cellulose digestion by equine microorganisms and enhances gross energy digestion of feed. An eastern European report suggests that lucerne hay may have a protective value in the development of glandular inflammation and may encourage white cell (lymphocyte) and red cell (erythrocyte) production in foals (Romić 1978). Pelleted lucerne meal is a better source of nutrients than grass hay or sun-cured lucerne (except for vitamin D₂), and the oxalates it contains do not hamper its calcium or magnesium digestibility.

The drying takes place at very high temperatures, sufficient to inactivate mould spores. This will extend shelf-life, inhibit mould growth during storage and so reduce the likelihood of adverse respiratory responses. Pelleting of lucerne leads to increased consumption and its relative bulkiness prevents a rapid accumulation of highly fermentable starch in the stomach, while allowing the penetration of it by gastric juices, ensuring a fall in gastric pH. Horses fed dried lucerne seem to produce better hoof horn than those receiving grass hay. The explanation may be that lucerne provides considerably more sulphur-containing amino acids and calcium than is provided by grass hay.

Dried lucerne leaves contain several green pigments in addition to chlorophyll. One of these, pheophorbide- α , has been shown to cause photosensitization, expressed as skin lesions, of albino rats. Exposure to light in the visible range is sufficient to cause lesions in rats and other white animals, given a lucerne-leaf-protein concentrate. The pheophorbide- α is formed by breakdown of chlorophyll under the influence of chlorophyllase, during processing. There is a higher activity of this enzyme in legume forages than in grass, which may account for an absence of dermatitis associated with green grass products.

Carrots (*Daucus carota*)

Carrot roots are very palatable to horses and contain no undesirable chemicals, as long as they have not been moulded. Horses not accustomed to their consumption may bolt them, developing choke, and in that case it is desirable they should be sliced. They have a dry matter content of 110–130 g/kg and an energy value rather similar to that of oats per unit DM. The orange-coloured varieties are rich in β -carotene, containing 100–140 mg carotene/kg, 85% of which is present as the β -isomer. This is partially converted to vitamin A by the horse. Large intakes of

carrots, and therefore of the provitamin, can cause intoxication in the human. β -carotene has been demonstrated by some workers to improve fertility in mares deprived of a dietary source (Ahlsweide & Konermann 1980; Ferraro & Cote 1984; van der Holst *et al.* 1984) (see Chapter 4).

Potatoes (*Solanum tuberosum*)

Small (chat) and damaged potatoes are sometimes fed to livestock. However, green potatoes and sprouted potatoes contain the alkaloid solanin, which is extremely hazardous to horses. Damaged potatoes and those commencing to decompose are equally dangerous and there have been several reported fatalities. Small potatoes may also cause choke. It is not recommended that waste potato feeding be practised with horses. Moreover, Meyer *et al.* (1995) reported that the pre-ileal digestibility of potatoes or cassava was less than 10% compared with 80–90% for oat starch.

Citrus pulp (*Citrus* spp. and *Ananassa sativa*)

Juice extraction from oranges, lemons, tangerines, limes and pineapples (*Ananassa sativa*) leaves a residue of pulp (peel, pith and seed) that is dried and pelleted, frequently after adding limestone to neutralize the acid and to aid the removal of moisture (average calcium content then is 12 g/kg). The product is low in protein and phosphorus, rich in fermentable fibre (mostly pectins), clean and a useful addition to horse feeds, although the palatability may be variable. The content of oil may vary from 10–70 g/kg and that of crude fibre averages 130 g/kg. A high fibre level tends to indicate the presence of citrus seeds that contain relatively high concentrations of limonin, a metabolically interesting compound in human medicine that is toxic in high concentrations to pigs and poultry. The toxic threshold in horses has not been determined, but inclusion rates of the pulp in concentrates of up to 50 g/kg should be quite safe where seeds are present, and of up to 150 g/kg where they are scarce or absent. Ammoniated pulp is not recommended.

Olive pulp (*Olea europaea*, *O. sativa*)

The pulp and skin of olive fruits, following oil expression or extraction, is dried and pelleted. Where expression is used the pulp may contain 260 g crude fibre/kg and 100–180 g oil/kg which can be rancid and lead to increased vitamin E demands. For extraction, carbon disulphide, trichlorethylene or benzene is used and unsatisfactory solvent residue may be present. The product is equivalent to a poor roughage. Pressed cake containing the seeds causes digestive problems, and the digestibility generally is only moderate. The calcium and phosphorus contents average 10 and 2 g/kg, respectively; the protein (100 g/kg) is of poor quality and low digestibility. Some batches have been of recognizable value for horses when included in the diet up to 100 g/kg, but the variability of the product does not lend it to any general recommendation.

Cottonseed hulls (*Gossypium* spp.)

The hulls of cottonseeds, to which some fibres are attached, are free from gossypol and result from the decortication of the seed. Cottonseed hulls yield a similar feed intake and growth rate to that of coastal Bermuda grass when each is used as the single roughage source in rations for young horses (Heusner *et al.* 2001).

Soya hulls (*Glycine max*)

Soya hulls are rich in pectin and other soluble fibres (g/kg: 131CP, 606 NDF, 437 ADF). Booth *et al.* (2003) replaced up to 75% of alfalfa-brome grass hay by soya hulls without loss of acceptability. The consequential increase in dietary soluble fibre caused increases in total caecal VFA production and the proportion of propionate in the VFA, but a decrease in the proportion of butyrate and in caecal pH from 7.0 to 6.45.

Cassava (manioc) (*Manihot esculenta*)

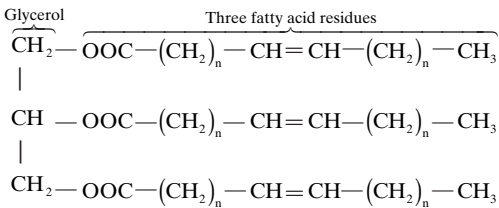
Cassava is a woody herbaceous shrub of tropical and subtropical areas. The tuberous roots provide a human and livestock food that is inferior to cereals and low in protein. The roots contain a glucoside, linamarin, which liberates hydrocyanic acid (HCN) by enzymic hydrolysis. A dried root meal, from which some of the starch has usually been extracted, is widely available in many Western countries. This should contain a minimum of 620 g starch/kg, a maximum of 50 g fibre/kg and 30 g silica/kg (mostly from adherent soil). It also contains a residue of the glycoside and HCN so that its value for horses is limited.

FAT SUPPLEMENTS

There is increasing interest in supplementing horse feeds with edible fats or edible oils (see Chapter 9). Good sources of well-digested and edible oils and fats have a calming effect on excitable horses. This effect may be imparted by the lecithin that oils contain (Holland *et al.* 1996). Choline, which is found in lecithin, is a component of the neurotransmitter acetylcholine found in sympathetic and parasympathetic ganglia and at the myoneural junction.

Where oils and fats are by-products of human food production, or of industrial processes, the quality of the fat or oil can be extremely variable. In order to establish the feeding value of a parcel, the fatty acid composition is frequently sought. This is generally inadequate and therefore it is opportune at this point to give some description of fats and of analytical procedures used in their definition.

Crude fat consists of triacylglycerols (three fatty acid residues combined with glycerol, see Fig. 5.2); long chain (aliphatic) alcohols, as found in waxes; choline; phosphate; sterols (cyclic alcohols), such as cholesterol; moisture; and oxidized,



Under severe heating of fats, commonly with unsaturated fatty acids present, these monomers may combine by rearranging the linkages, forming new carbon-carbon double bonds, resulting in indigestible dimers and trimers.

Polymerization of fats

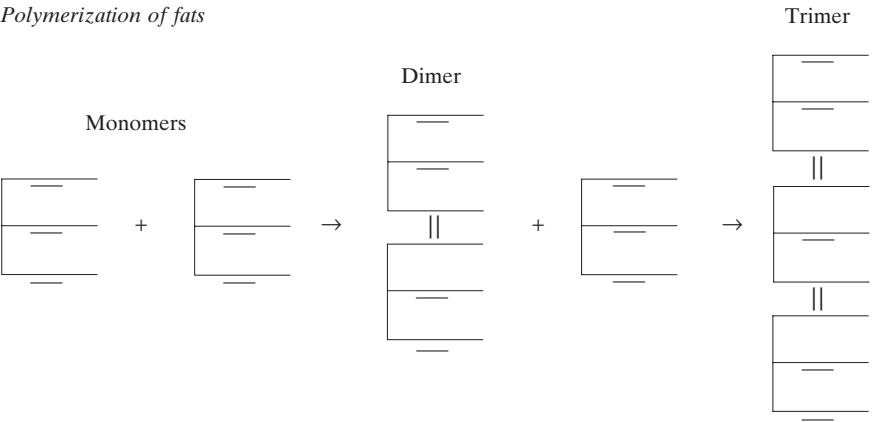


Fig. 5.2 A triacylglycerol molecule (simplified to show one unsaturated bond in each fatty acid).

Table 5.7 Composition of typical feed fat and desirable limits (g/kg).

	Typical	Desirable maximum
Moisture and other impurities	8	10
Unsaponifiables	20	30
Glycerol	25	
Total fatty acid	870	
Free fatty acid	60	60
Oxidized fatty acid	20	25
Oxidized, polymerized fatty acid	77	0
Polythene	—	0.2
Total	1080	

polymerized and cyclized fatty acids. A typical feed fat (material soluble in a fat-solvent, e.g. ethanol, petroleum and diethyl ether) may have the composition given in Table 5.7. Table 5.8 gives the characteristics of the gas-liquid chromatographic (GLC) fatty acid profile of fats suitable as horse feed supplements.

Only the glycerol and fatty acids have a reliable energy value. To determine the fatty acid composition of fat it is hydrolysed by saponification, yielding the sodium

Table 5.8 Desirable GLC profile of fatty acids in fats used as supplements (g/kg fat).

Saturated fatty acids	300
stearic acid (C18:0)	100 maximum
palmitic acid (C16:0)	200
Monounsaturated fatty acids	500–600
palmitoleic acid, oleic acid (C16:1, C18:1)	500–600
Polyunsaturated fatty acids	250
linoleic acid, linolenic acid (C18:2, C18:3)	200 maximum
arachidonic acid plus others, e.g. eicosapentaenoic acid (all in animal fats) (C20.4+)	50 maximum
Percentage total fatty acid content by internal marker	800 minimum

or potassium salts of fatty acids and free glycerol. The unsaponifiables (insoluble in water, but soluble in fat-solvents) include hydrocarbons, aliphatic alcohols, cholesterol and phytosterols. These unsaponifiables, together with the high-molecular-weight polymerized and cyclized fatty acids are not eluted from a column with the fatty acids during GLC analysis and so *they are not recorded in the standard fatty acid profile of the fat*. The value for each fatty acid in this analysis is given as a percentage of the sum total of the fatty acids eluted and the values can therefore appear quite normal for fats containing damaged nonelutable fatty acids, as the total fatty acids will summate to 100%. Note that this is the percentage of the total eluted fatty acids and not of the total fat supplement purchased. The author has examined feed fats containing less than 400 g utilizable fatty acids/kg fat (<200 g/kg fat supplement, for most fats are sold on an inert base). If an internal marker of an unusual fatty acid, e.g. an odd-numbered carbon fatty acid, such as C17, is included in the analysis, an estimate may be made of the true proportion of the utilizable fatty acids in the total fat.

Polymerization and cyclization of fatty acids occurs during excessive heating of fat (thermal decomposition) (see Fig. 5.2). Fat that is heated in air also undergoes oxidative decomposition, and the extent of this tends to be correlated with the extent of polymerization. An indication of the existence of oxidation can sometimes be obtained by measuring the peroxide value of the fat. This is a measure of the oxidation of the double bonds in unsaturated fatty acids and it is likely to be more extensive in polyunsaturated oils unprotected by antioxidants. Values in excess of 20 mEq/kg oil must be suspicious. Low peroxide values, on the other hand, do not indicate a lack of damage, as peroxidation is a primary stage in the deterioration of fat quality. Secondary decomposition includes the formation of carbonyl-, hydroxy- and epoxy-derivatives, which are also valueless. The fat may have become hydrolysed, liberating free fatty acids, hence the value of determining free fatty acids in raw material that is not intended to contain such acids. (Hydrolysed edible fat containing 50% free fatty acids is also marketed. This is a very satisfactory product.) Saturated fatty acids can become rancid only following hydrolysis, as in butter, indicating deterioration by ketone formation.

The importance of using an internal marker in fatty acid analysis should be apparent. If such an analysis gives a value of 18% for, say, the C16 fatty acid as a

percentage of eluted fatty acids in a product, which is sold as 50% fat on a nonfat base, then the actual amount of the C16 fatty acid present in the product is only 3.6%, where there are only 400g utilizable fatty acids/kg in the product referred to above. Typical bases used for preparing fat premixes include palm kernel extractions, ground straw, wood flour and vermiculite, a hydrous silicate.

Damaged fatty acids in fats may have a negative value, precipitating a vitamin E deficiency and may cause digestive upsets and other problems. Waste oils that are notorious for causing problems of these sorts are acid oil distillation residues, especially fish acid distillation residues. Other undesirable by-products are 'roller oil' from the steel and tin plating industry, oxidized cod oil, obtained from tanned hides, and recovered vegetable oil (RVO) from food frying outlets. Waste fats can be contaminated with heavy metals, pesticides or polythene. The rate of deterioration of a fat will be accelerated by contamination with, for instance, copper and retarded by the presence of antioxidants (synthetic or natural). Most seed oils are highly unsaturated, but if fresh are generally rich sources of the antioxidant α -tocopherol (e.g. soya-bean oil). Marine oils will deteriorate more rapidly than, for example, coconut oil, as a consequence of the difference in degree of unsaturation.

High-quality fats, whether highly saturated or polyunsaturated, have a valuable role to play in the nutrition of stabled horses and it is imperative that the quality of the fat supplement is assured for this value to be realized. Edible oil probably reduces the rate of flow of ingesta from the stomach, as the addition of 10% maize oil dramatically decreases the glycaemic index of sweetfeed (Pagan *et al.* 1999a). Moreover, according to some evidence, high-quality vegetable oil and animal fat do not adversely affect fibre digestibility (Bush *et al.* 2001; Kronfeld *et al.* 2002) and they are fully digestible, yielding a DE value of 38MJ/kg. Nevertheless, they may depress protein digestibility (Kronfeld *et al.* 2002).

On the other hand, with high inclusion rates, Jansen *et al.* (2000) reported that 150g soya-bean oil/kg diet, substituted for isoenergetic amounts of corn starch, reduced apparent digestibilities of crude fibre, NDF and ADF by 8.0%, 6.2% and 8.3% respectively. Jansen *et al.* (2002) subsequently concluded that this excessive inclusion rate increases the amount of fat entering the large intestine, and its specific inhibitory effect on microbial fermentation causes a depression in fibre digestibility (Table 5.9). Evidence also indicates that no fat absorption occurs in the large intestine of horses (Swinney *et al.* 1995), so that fat digestibility would be depressed,

Table 5.9 The effect of crude fat and nitrogen-free extract composition of complete diets on apparent total intestinal tract digestibility of structural carbohydrate % of intake (Jansen *et al.* 2002).

	Soya oil	Corn starch	Glucose
Crude fat, g/kg DM	148	20	20
NFE, g/kg DM	493	668	646
Crude fibre digestibility, %	56.6	70.7	71.0
NDF digestibility, %	64.4	73.8	74.4
ADF digestibility, %	55.0	68.3	68.4
Cellulose digestibility, %	55.6	72.5	72.1

imposing a practical ceiling on the inclusion rate. Fat supplementation up to 100 g/kg diet is well utilized. It increases the energy value of feeds, eases pelleting of fibrous feedstuffs and reduces product dustiness.

Polyunsaturated fatty acids

The constituent oils of most seeds and cereal grains are rich in n-6 polyunsaturated fatty acids. The sources include corn oil, sunflower seed oil, and, more recently, and similar to sunflower, grape seed oil and even oat oil (Taylor *et al.* 2003), whereas only a few, including soya oil, linseed oil and rapeseed oil, contain any significant amounts of n-3 polyunsaturated fatty acids. These two series are differentiated by the position of the first double bond in the carbon chain, which, in the case of the n-6 series, is six carbons from the terminal methyl group. Fish oils are rich in the higher (i.e. longer and more unsaturated chains) members of the n-3 group. Linoleic acid (C18 n-2) of the n-6 series in seed oils is elongated and desaturated during metabolism to arachidonic acid (C20:4 n-6) which is the precursor of the inflammatory eicosanoid prostaglandins.

Horses in hard work receive a large cereal-based diet, that is, one in which the fat is rich in n-6 polyunsaturates. On the other hand, the grazing and browsing horse receives a diet in which the oils are derived from leaves, which by happy chance contain a much higher proportion of the lower members of the n-3 series. Competition is afforded by these n-3 nutrients during the elongation, desaturation and cyclo-oxygenase metabolism of the n-6 series, and thus the synthesis of inflammatory intermediates is partially suppressed.

In fact there is much evidence in the human field that cod liver oil reduces inflammatory reactions in joint disease. Comparable evidence is coming forward that similar responses may be expected in horses. Recently, techniques have been developed to retain an omega 6:omega 3 ratio of 6:1 in soya oil, and, when added to the diet of strenuously exercised horses at the rate of 100 g/kg, cf corn oil, it was shown to reduce the inflammatory response, measured as the rise in blood fibrinogen, an acute-phase protein (Wilson *et al.* 2003). Thus, working horses are subject to inflammatory ailments and so supplementation of their diet with oils rich in n-3 fatty acids may reduce the extent of painful leg ailments. A word of caution, however: fish oil n-3 fatty acids are more unsaturated than oils of the n-6 series and any increased use of the n-3 sources should be accompanied by an increase in vitamin E supplementation.

PROTEIN CONCENTRATES

Vegetable proteins

The richest sources of vegetable protein fed to horses and ponies are oil-seed residues, but other sources include peas, beans, yeast and, in the future, possibly new sources of microbial protein and, finally, high-quality dried forages, particularly lucerne meal (alfalfa). Soya beans, linseed, cottonseed and, to some extent,

sunflower seed after processing are widely used. Groundnuts cannot be recommended because of their frequent contamination with a toxin (aflatoxin, also found in some other feedstuffs) of the mould *Aspergillus flavus* to which the horse is relatively sensitive.

Two alternative procedures are adopted for the extraction of oil from oil seeds, both of which may be preceded by the removal of a thick coat by a process known as decortication, as practised for cottonseed and sunflower seed. Uncorticated meals contain very much more fibre. Where oil is removed by pressure, this is preceded by cooking at up to 104°C for 15–20 min, after which the temperature is raised briefly to 110–115°C. Then pressure is achieved by passing the seeds through a horizontal perforated cylinder, in which a screw revolves and the oil is partially pressed out, leaving a residue containing perhaps 35 g fat/kg. Expeller cakes, therefore, have the advantage of incorporating more fat than meals derived from the more efficient chemical-extraction process. However, the temperatures achieved during compression can damage the protein, which generally has a lower biological value (see Glossary) than that resulting from solvent extraction. In this latter process only material with less than 350 g oil/kg is suitable so that the feeds are first subjected to a modified screw press, less extreme in its effects than in the expeller process. The seeds are then flaked and the solvent, usually hexane, is allowed to percolate through, effectively removing the oil. The solvent residues are evaporated by heating or toasting, which also benefits some meals by destroying natural toxins.

Oil-seed meals are much richer sources of protein than are cereals and their balance of amino acids is superior. However, linseed meal is a poorer source of lysine than is soya, considered the best quality of these proteins. Sunflower seeds are rich in the sulphur amino acids, cystine and methionine, although it is rare for horse diets to be limiting in respect of these amino acids. Oil-seed meals are also relatively reliable sources of some of the B vitamins and of phosphorus, but contain little calcium.

Soya-bean meal (Glycine max)

Raw soya beans contain allergenic, goitrogenic and anticoagulant factors in addition to protease inhibitors. The correct toasting and cooking of the beans, as in micronization and well-regulated oil-extraction procedures, destroy these factors without detracting from the protein quality (in fact amino acid value is radically influenced by variation in heat treatment, see 'Cottonseed meal', below). Reliably cooked products, therefore, may be used as the sole source of a supplementary protein in horse feeding.

Standard hexane-extracted soya-bean meal contains 440 g crude protein/kg. Dehulled meals of uniformly high quality containing 480–490 g crude protein are also of general commercial availability. Both these meals contain less than 10 g oil/kg. Full-fat soya flour and cooked soya flakes are much more costly, but the latter is widely used in coarse mixes and both contain 180–190 g fat and 360–400 g of crude protein/kg. The precise composition varies with the crude-fibre level, which ranges from 15 to 55 g/kg.

Linseeds and linseed meal (Linum usitatissimum)

Linseeds are unique in so far as they contain a relatively indigestible mucilage at concentrations of between 30 and 100 g/kg. This can absorb large amounts of water, producing a thick soup during the traditional cooking of linseed, and its lubricating action regulates faecal excretion and sometimes overcomes constipation without causing looseness. The cooking of linseed also destroys the enzyme linase, which, after soaking, would otherwise release HCN from a glycoside in the seeds, in the presence of water, so poisoning the horse. This action implies that the seeds should be added to boiling water rather than to cold water and then boiled, otherwise some enzymatic activity may be initiated. However, HCN is volatile and a proportion of any already present will be driven off by subsequent boiling. Cooked linseed mashs are highly regarded to improve coat condition of horses for sale. Linseeds should not be fed dry because of their water-absorbing propensity, although the contained linase would be rapidly inactivated by the stomach's secretions.

The low-temperature removal of oil during the production of linseed meal implies that the product may be toxic if fed as a gruel, linase may not be inactivated and HCN can be evolved. In comparison, oil removal by the expeller process normally yields sufficient heat to make a safe cake, whether it be fed wet or dry. UK laws stipulate that linseed cake or meal must contain less than 350 mg HCN/kg, although this takes no account of any linase activity that may be present.

Coconut meals (copra) (Cocos nucifera)

The dried flesh of the coconut contains variable amounts of oil (50–70 g/kg) depending on the efficiency of extraction. The oil is composed of highly saturated, medium-chain fatty acids and so is less subject to rancid fat development than many other vegetable oils. The meal is prone to absorb moisture and mould unless lengthy storage is avoided and an environment of low humidity is achieved. It contains a similar amount of protein to that of peas, but of lower biological value (see Glossary). The meal typically contains 220 g protein, only 6 g lysine and 125 g crude fibre/kg. It is palatable and contains no toxic factors.

Cottonseed meal (Gossypium spp.)

Cottonseed meal is a cake by-product of cotton and oil production and it tends to be dry and dusty, having a somewhat costive (constipating) action. Undecorticated, the cake contains 200–250 g crude fibre/kg. If the hulls are separated from the kernels the fibre content is halved. The oil is removed either by hydraulic expeller or solvent extraction methods. Glands within the seed embryo contain a toxic yellow pigment, gossypol, which may be at a concentration in raw cottonseeds of between 0.3 and 20 g/kg DM. Heating during processing partly inactivates the toxin in the raw material, but at excessive temperatures gossypol binds lysine, depressing protein quality. The binding process partially inactivates the toxin. However, even the

bound form is reported to reduce intestinal iron absorption, partly counteracted by further iron supplementation. Free gossypol may be at a concentration of 1 g/kg in expeller meals and 5 g/kg in solvent-extracted meals. Mixed feeds containing more than 60 mg of free gossypol/kg are unsatisfactory for horses. Therefore only products of glandless varieties are suitable for horse feeding and these are scarce in Europe. Cyclopropene fatty acids are also present in cotton seeds, but at lower concentrations in glandless varieties, although the amount is proportional to the oil residue in the meal. Thus, extracted meals of glandless varieties should be sought, notwithstanding the limitation placed by the Feeding Stuffs Regulations on the gossypol content of meals sold in the UK to 1.2 g/kg. Good quality cottonseed meals are palatable and can contain 40% protein and 15 g lysine/kg.

Moreover, Gibbs *et al.* (1996) reported that the precaecal N digestibility of a sample of solvent-extracted cottonseed meal they examined amounted to 81.2% in comparison with only 57.1% for their sample of soya-bean meal. The difference may have relied upon excessive, or inadequate, heat treatment of the soya. Although the N digestibilities over the entire GI tract were similar for the two samples, the amino acid value of dietary proteins depends on the extent to which precaecal digestion occurs (see Chapter 1).

Sunflower-seed meal (Helianthus annuus)

Sunflowers are grown for the oil in their seeds. The oil content of the meal residue is greater where the oil has been removed by hydraulic pressure rather than by extraction. However, the oil in the residue is unstable. The fibre is poorly utilized and the fibre content of the meal is much lower where the seeds have been partially dehulled (decorticated) and so it ranges from 50 to as much as 350 g/kg. The protein is of high quality, rich in sulphur-containing amino acids (double that of soya protein), but variable in amount (from 240 g protein/kg in expeller cake to 500 g/kg in some samples of dehulled extracted meal). The methionine and lysine contents of the meal are 5–14 and 9–22 g/kg, respectively, and the meal is a good protein source for horses, containing no significant undesirable substances, but it may be unpalatable in large quantities.

Palm-kernel meal (Elaeis guineensis)

Expeller and extracted meals are available, but they are not widely used for horse feeding owing to low palatability. The protein and crude fibre contents are typically both 160 g/kg; the protein is relatively impoverished of lysine (40 g/kg protein). Nevertheless, the meals are free from undesirable chemicals.

Groundnut meal (peanut meal) (Arachis hypogaea)

Groundnut or peanut meal is produced following oil removal by expeller and extraction processes. The former process leaves a larger residue of the polyunsatu-

rated oil. The protein content of the meal is slightly higher than that of standard 44% soya-bean meal and averages 470–480 g/kg. Groundnut meal is very palatable and contains no undesirable substances apart from frequent contamination with the hepatotoxin, aflatoxin, derived from the mould *Aspergillus flavus*. The horse is subject to severe aflatoxicosis, and, owing to a history of serious damage among livestock, the use of this by-product in Europe has been severely curtailed by EU legislation. If this problem is resolved then groundnut meal could make a useful contribution to equine nutrition.

Rapeseed meal (Brassica napus, B. campestris)

Rape is a member of the genus *Brassica* in the family Cruciferae and it is grown for the oil in its seeds. There is a large world production of rapeseed; varieties from two species are grown – *Brassica napus* and *B. campestris*. Western European production has increased rapidly owing to encouragement by the EU. A drawback to rapeseed in the past was the content of erucic acid in the residual oil and of glucosinolates in the meal. Glucosinolates, present in the unheated rapeseed, and widely distributed among the Cruciferae, are goitrogenic when hydrolysed during digestion by the enzyme myrosinase, present both in unheated rapeseed and in gut microorganisms. The active goitrogens released are isothiocyanates and oxazolidinethiones (goitrin) (see ‘Goitrogens’, this chapter). Although the protein quality of rapeseed meal is good and although heat treatment decreases the hazard by destroying the myrosinase, the intestinal enzyme can still release quantities of the thyroactive substances and so only small amounts of meal of unknown origin are suitable for feeding to horses and many other animals.

This problem led Canadian plant breeders to select varieties of *B. campestris* low in both erucic acid and glucosinolates (less than 3 g/kg seed), known as ‘double-low’ varieties and sold as canola meal. Comparable varieties are widely available in Europe, leading to their routine cultivation. The better varieties are a good source of protein for horses, but use should be restricted to 200 g/kg concentrates. The protein content of the meal ranges from 340 to 390 g/kg and the protein contains 60–64 g lysine/kg, and so the meal may replace soya on an equivalent protein basis. Several rapeseed varieties contain tannins, or polyphenols (averaging 30 g/kg seed) that reduce digestibility, limiting their usefulness and possibly contributing to a slightly lower protein value for the meal in comparison with that of soya, according to several reports (reviewed by Aherne & Kennelly 1983). A further discussion of these toxins is given later in this chapter (see ‘Condensed tannins’).

Lupin-seed meal (Lupinus albus, L. angustifolius, L. luteus)

Lupin-seed meal has two disadvantages for horse feeding. There are three species grown that have white, blue and yellow flowers and a number of varieties within each species. These are variably sweet or bitter. The bitter varieties contain significant amounts of toxic alkaloids and the ether extract content is variable

(40–120 g/kg) and prone to rancidity. The crude fibre content is high and variable (80–160 g/kg), but relatively well utilized by the horse, and the protein and lysine contents vary from 250 to over 400 g/kg and from 14 to 23 g/kg, respectively. The seeds are small and must be ground, or crushed, for use. Only the sweet varieties should be used and limited to 50 g/kg concentrate.

Sesame-seed meal (Sesamum indicum, S. orientale)

The seed oil of sesame is a source of unstable polyunsaturated fatty acids and so only extracted, and not expeller, meal should be used, which contains 440 g crude protein/kg, rich in methionine but deficient in lysine. The meal is also rich in P, mainly present as phytates. The husks contain oxalates so that only decorticated meal is suitable.

Peas (Pisum sativum, P. arvense), white- and purple-flowered

Peas contain less protein than do field beans, but the biological value (see Glossary) to horses is equivalent to that of soya protein. Legume seeds generally contain antinutritive factors, although the content in modern pea varieties is low. Some older varieties may contain trypsin inhibitor and phytohaemagglutinins, which are partially inactivated by heating. Autoclaving for 5 min at 121°C completely destroys pea trypsin inhibitors and haemagglutinins. Trypsin inhibitors are stable at 80°C or below. Soaking for 18 hours removes 65% of the haemagglutinin activity. The trypsin inhibitor content of peas is similar to that in field beans, but only 10% of that in soya-beans. Peas also contain chymotrypsin and amylase inhibitors, although the content of the latter is very low and destruction by heating is akin to that for trypsin inhibitor.

The oxalate content of peas is about 7 g/kg, phytic acid 5–8 g/kg and tannins (polyphenolic compounds) 0.2–0.4 g/kg. Tannins, mostly in the testa (seed coat), bind to enzymes and other proteins forming insoluble complexes, reducing digestibility. Extended cooking is a good insurance (see ‘Natural and contaminant toxicants in feeds’, this chapter). The expansion (the extent of cooking varies among machine types) and micronization processes adopted for peas included in coarse mixtures and other compounded feeds are adequate to achieve a significant improvement in nutritive value. Nevertheless, peas are a very useful protein source for horses.

Field beans (Vicia faba), winter beans (horse) and spring beans (tick and horse)

Within the bean family (Leguminosae or Fabaceae) two genera, *Vicia* and *Phaseolus*, grow throughout the world, and many are important food crops. Winter and spring varieties of field (horse) beans grown in the UK are members of the species *Vicia faba*, all safe for feeding to horses, especially after cooking. The winter field (horse) bean contains on average 230 g crude protein and 78 g crude fibre/kg,

whereas the spring bean contains 270g crude protein and 68g crude fibre/kg. Amounts of fat in both varieties are low – about 13 g/kg; like most other seeds they are rich in phosphorus but poor in calcium and manganese. Field-bean protein is of high quality as it is a valuable source of lysine. The bean is normally cracked, kibbled or coarsely ground, but may be given whole to adult horses with sound teeth. The water-soluble polyphenolic condensed tannins in the hulls interact with both dietary and endogenous proteins in the intestines, increasing the faecal losses of both these protein sources. High dietary concentrations of condensed tannins depress appetite; however, the extent to which this occurs in the horse has not been determined.

Phaseolus and some other legume species

There are importations of beans belonging to the genus *Phaseolus*, especially the lima (navy) bean (*P. lunatus*) and the kidney (haricot, pinto and yelloweye) bean, (*P. vulgaris*). Kidney beans are normally refused by horses unless cooked; if force-fed they will cause colic. These beans must all be cooked (wet heat) before feeding because they contain several toxic factors, including antiproteases and lectins, which will cause diarrhoea, and many beans of the genus *Phaseolus* also contain a cyanogenetic glycoside identical to that in linseed. Other beans available in parts of the world include the hyacinth bean or lablab (*Dolichos lablab*), horse gram (*D. biflorus*), green and black grams (*P. aureus*, *P. mungo*) and chick pea (*Cicer arietinum*), which are widely used in Asia, and lentils (*Lens* spp.) of which India is the chief producer. One lentil type is said to induce staleness in racehorses and hunters if given in excess. The Indian or grass pea (*Lathyrus sativus*), at one time imported as an animal feed, and some other members of the same genus, cause lathyrism (see ‘Lathyrism caused by $\beta(\gamma$ -L-glutamyl) aminopropionitrile and L- α , γ -diamino butyric acid’, this chapter). The horse is particularly, and characteristically, affected. If beans and peas of unknown origin are used to feed horses, extended cooking will provide a measure of safety from many of the toxins.

Lentils, split peas and red dahl (Lens esculenta, L. culinaris)

Lentils, split peas and red dahl are important and palatable legume crops, producing seeds containing protein of good quality (protein 260g/kg, lysine 70g/kg protein), for human consumption. They may contain small amounts of trypsin inhibitors and haemagglutinins and as they are in demand for human use those batches available as livestock feed may be contaminated with moulds and mycotoxins. Such batches should be avoided. Lentil bran may be obtained as a by-product of lentil preparation for human consumption.

Animal proteins

There are only two high-quality animal protein sources suitable as horse feeds – white fish-meal and milk-protein products. They are reserved almost entirely for

foals, either in creep supplements or as milk replacers. Small amounts are occasionally given to adult horses in poor condition, but large amounts of dried skimmed milk may cause diarrhoea, owing to the presence of lactose (see Chapter 1).

Fish-meals

Two types of fish-meal are recognized under British law, of which the first is a product from the drying and grinding of fish, or fish waste, of a variety of species. The second, marketed as white fish-meal, is a product containing not more than 4% salt and obtained by drying and grinding white fish or the waste of white fish to which no other matter has been added. This is a high-quality protein source because it contains abundant lysine, and it is suitable for, but not essential in, the diet of young foals. It is rich in minerals (about 80 g calcium and 35 g phosphorus/kg), trace elements (especially manganese, iron and iodine) and several water-soluble vitamins, including vitamin B₁₂. This vitamin is found naturally only in animal products and bacteria. The dietary requirement of the young weaned foal for the vitamin can be met by the inclusion of fish-meal, or synthetic sources, in its diet. The suckling foal should, however, receive ample in the dam's milk. About 5 or 10% of white fish-meal in a creep feed, or milk replacer, is quite satisfactory for foals. During processing the fish waste is dried by one of two procedures. The first and more desirable one is steam drying, either under reduced pressure or with no vacuum applied. The other procedure is based on flame drying, when the temperatures achieved may decrease the digestibility of the protein and decrease the content of available lysine.

Fish-meals of unknown origin may be contaminated with pathogenic organisms, in particular with *Salmonella* species or other enteric organisms that cause diarrhoea. Good quality white fish-meal, however, should be a safe feed. Meat meals, meat and bone meals and unsterilized bone flour should on no account be used for feeding to horses because many samples are contaminated with *Salmonella* and other transmissible pathogens, including bovine spongiform encephalopathy (BSE), or are shipped in contaminated bags. The problem of ridding stock of infection, once contracted, is considerable.

Cow's milk

Where liquid milk is used for feeding to orphan foals, it should be diluted with 15–20% of clean water and given in small amounts in as many meals as is practically convenient. Liquid cow's milk, on average, contains 125 g DM, 37 g fat, 33 g protein and 47 g lactose/kg. It contains little magnesium and is deficient in iron, a source of which should be provided for the young foal (at birth the foal's liver acts as a reservoir of iron for the neonatal foal). Whole milk is rich in vitamin A and provides useful quantities of vitamin B₁₂, thiamin and riboflavin. Milk proteins contain abundant lysine.

Dried skimmed milk is widely available and sold commercially as a component of milk replacers and horse supplements. As its name implies, it contains very little fat

and therefore practically none of the fat-soluble vitamins. However, the protein quality approaches that of the liquid product if drying has been carried out by the spray process. Roller drying subjects milk to higher temperatures which results in some loss of lysine availability, and in large quantities this product can cause diarrhoea. Any significant quantity of dried skimmed milk should not be fed to horses more than three years old, owing to their deficiency in the enzyme lactase (β -galactosidase) that digests lactose.

Spray-dried skimmed milk is a useful supplement to feeds of young foals where the mother is providing inadequate quantities of milk to sustain normal growth. Concentrations of 10–15% in the dry diet have proved satisfactory. On the other hand, its use in creep feed for foals approaching weaning may be less satisfactory if the main objective is to encourage the development of a faculty for the digestion of horse feeds to be given after weaning. Thus, satisfactory creep feeds for use in normal circumstances can be provided as nutrient-rich stud nuts.

Single-cell proteins

Yeasts (Saccharomyces cerevisiae, S. carlsbergensis)

Yeasts contain protein of good amino-acid balance. However, a greater proportion of the N in single-cell organisms (bacteria in particular and yeasts) compared with plant cells is composed of nucleic acids (50–120 g/kg DM in yeasts and 80–160 g/kg DM in bacteria). While some of the purine and pyrimidine bases in these acids can be used for nucleic acid biosynthesis, large amounts of uric acid, the end product of nucleic acid catabolism, are excreted in the urine. Dietary inclusion rates of up to 50–75 g/kg feed are economically feasible only for foals.

The crude protein and fat contents range, respectively, from 400 to 450 g/kg and 25 to 55 g/kg. Yeasts are readily digested and are a rich source of B-group vitamins with the exception of vitamin B₁₂. This vitamin is synthesized almost exclusively by bacteria. The live yeast culture has been demonstrated to promote fibre digestion and growth rate in young horses when included in the diet at a concentration of only 1 kg/tonne (see 'Probiotics' below).

Bacterial cultures

The crude protein and fat contents of bacterial cultures range, respectively, from 340–720 g/kg and 20–210 g/kg, but their use as a dietary protein source is not generally justified (see 'Probiotics' below).

PREBIOTICS

Substances added to the diet that preferentially stimulate the growth of beneficial microorganisms in the hind-gut have been termed *prebiotics*. These include the

rapidly fermented fibres fructo-, galacto- and mannanoligosaccharides (also see 'Laminitis control', Chapter 11). When 10g/day of mannanoligosaccharide was given to pregnant mares from 56 days before foaling, there was a marginal increase in colostrum IgG, IgM and IgA concentrations (Spearman *et al.* 2003). In accord with this, foals maintained on pasture containing mixed legumes and grass and given a supplementary diet high in fat and fibre, cf. high sugar and starch, both before and after weaning, produced higher plasma IgA, IgG and α -tocopherol concentrations, a higher faecal pH and lower faecal butyric and valeric acid concentrations (Swanson *et al.* 2003). This observation is, nevertheless, surprising as pasture grass is variably rich in fructo-oligosaccharides.

PROBIOTICS

Over the past two decades there has been increasing interest in the inclusion of probiotics in equine diets. Probiotics, or substances 'for life', may be defined as live microorganisms and their products that, when administered in adequate amounts, confer a benefit to the health of the host, principally through their influence on the microbial flora of the GI tract. Under EU legislation only registered live culture strains may be used for feeding to animals. The microorganism strain must be identified according to a recognized international code, including: the deposit number of the strain; the number of colony-forming units (expressed as CFU/kg, measured by an acceptable method); the period during which the CFUs will remain present; and the characteristics of the microorganism that may have arisen during manufacture.

Fungi

The organisms that are predominately used in probiotic cultures are certain species of Gram-positive lactic-acid-producing bacteria and several fungal species. The fungi are commonly strains of *Saccharomyces cerevisiae* (baker's and brewer's yeast), *Aspergillus oryzae* and *Torulopsis* spp. Studies by Glade & Sist (1990), in which *A. oryzae* has been added to *in vitro* equine caecal cultures, indicate that it does not alter fermentation products or pH. The consumption of live cultures of *S. cerevisiae* (Collection No. NCYC 1026) (10–20g/day per horse of a culture of 10^9 CFU/g), especially by young growing horses, has been demonstrated to stimulate hind-gut microbial growth, to improve dietary fibre fermentation (and so apparent digestibilities of pectins, hemicellulose, cellulose and crude protein). However, the apparent digestion of CP and Mg was increased in adult horses only when the culture was added to the diet at 5g/kg, but not at 10g/kg (Switzer *et al.* 2003). At intakes of 10–20g/day it has been shown to reduce endogenous (metabolic) faecal N loss, to increase N-retention and to increase plasma concentrations in foals of arginine, glutamine, glycine, isoleucine, leucine, methionine and valine, while concentrations of ammonia, hydroxyproline and 3-methyl-histidine were decreased (Glade & Sist 1990).

The addition of 10g live *S. cerevisiae* culture to the daily feed of horses, routinely given a starch overload, increased the viable yeast count in the hind-gut and modified the luminal pH, lactic acid and ammonia concentrations and molar percentages of acetate and butyrate, so that the undesirable changes wrought by the overload were limited (Medina *et al.* 2002). Yet changes in the composition of the digestive milieu frequently have an unclear interpretation. Similar additions have decreased the caecal acetate content resulting from both a high fibre and a high starch diet, but increased the caecal lactic acid content only with a high fibre diet, and not a high starch diet (Krusic *et al.* 2001).

A daily supplement of 10g dried live yeast (Hill & Gutsell 1998) increased the absorption of Ca and P, potentially as a result of increased hind-gut phytase activity, and it increased apparent digestibility of neutral detergent fibre (NDF), implying that yeast increased the extent of hemicellulose fermentation in the hind-gut, by stimulating the growth of cellulolytic bacteria (Medina *et al.* 2001). On the other hand, Yocum & Alston-Mills (2002) found that either *Kluyveromyces marxianus* or *S. cerevisiae* given daily from 14 days before foaling until day 42 of lactation reduced the lactose content of the milk.

One critical aspect of the subject is whether killed cultures of fungi, or bacteria, retain some of the useful attributes of live cultures. Evidence from ruminant research indicates that *S. cerevisiae* (Collection No. NCYC 1026) protects anaerobic rumen bacteria by increasing the rate of oxygen disappearance, indicating that viable cultures are necessary for this respiratory activity. Under EU legislation only registered culture strains may be used for live feeding to animals.

Bacteria

The bacterial species employed are Gram-positive organisms, and so they resist lysozyme. They ferment glucose and a variable number of other sugars with the formation of organic acids, principally lactic acid and acetic acid, and therefore they withstand a relatively low ambient pH. A culture of *Lactobacillus acidophilus* did not increase the degradation rate of lactate in the caecum (Booth *et al.* 2001) (NB: *L. acidophilus* ferments several sugars to form DL-lactic acid) and so it is questionable whether the use of this and related bacteria is justified for horses. The species are generally in the genera *Lactobacillus* and *Streptococcus*, although related species, including *Pediococcus* spp. are used. Certain other species employed may have disadvantages, including low viability in store or in the GI tract. The lactobacilli are probably all benign, whereas many *Streptococcus* species are parasitic in man and animals and some are highly pathogenic. Thus, cultures of the selected species must be pure.

The viability of species within the genera commonly used varies enormously among strains. Cultures should be preserved first by freeze-drying, the preferred method, in which water is removed by sublimation. With the object of extending shelf-life manufacturers frequently encapsulate organisms so that they may more readily withstand aggressive environments. The strain type selected for the horse should have been demonstrated to survive and multiply in the equine GI tract. This

assumes that viability is essential and, at this point, it is appropriate to remind ourselves of the characteristics that a useful culture of a strain should probably possess. It:

- should be incapable of causing disease or raising caecal lactic acid concentration;
- should contain 10^8 – 10^9 viable organisms/g after 12 months' storage;
- should have the correct host specificity, contain useful enzymes and certain antibacterial substances, and, if necessary, the strain should be capable of displacing lactic-acid bacteria occurring naturally in the intestine;
- should be Gram-positive and so resistant to gastric juice, both gastric juice's low pH and the enzymes it contains. Lactobacilli are generally resistant to the acidity encountered in the stomach, although the degree of resistance varies from strain to strain. One of the enzymes against which organisms must be resistant is lysozyme. Gram-positive lactic-acid bacteria have a greater resistance to lysis by this enzyme than Gram-negative coliforms. Gram-positive organisms are also more resistant to the adverse effects of freezing and freeze-drying;
- should be bile tolerant and tested on *Lactobacillus* selection (LBS) agar containing 0.15% oxgall. (Many bacteria entering the duodenum are destroyed by the bile secreted there. Bile salts lower surface tension and probably emulsify the lipids in the bacterial cell membrane.) Most strains of *L. bulgaricus* and *L. lactis* possess poor bile tolerance, despite their frequent inclusion in probiotic cultures.

Lactobacillus plantarum and *Bifidobacterium bifidum* are two species frequently used, but most strains fail to meet the above criteria adequately.

The possible functions of probiotics are:

- to act as a source of useful enzymes in the GI tract (Yogurt can increase lactose digestion in humans, even though the bacteria may not survive and/or grow in the intestines. The lactobacilli contain an intracellular β -galactosidase, which is thus protected during its passage through the stomach, but bile salts alter the membrane permeability of the bacterial cell, allowing ingress of lactose and access to the enzyme. If the bacterium grows in the intestine it will produce large additional amounts of the enzyme.);
- to inhibit the growth of intestinal pathogens (Many lactic acid bacteria have the ability to produce bacteriocins, antibiotic-like compounds that are active against closely related species. Enteric pathogens require their attachment to intestinal epithelial cells for pathogenicity. Precolonization and attachment to those cells by probiotic bacteria reduces this pathogenic potential which can occur at times of stress); and
- to assimilate cholesterol.

How these possible functions can lead to increased performance of the horse is unknown. The objective selection of fungal and bacterial species and strains in the future will depend on knowledge of how probiotics work.

Enzymes (bacterial and fungal)

Reference to enzymes was made under 'Bacteria' above. Enzymes are proteins and, therefore, can be denatured. Those contained within microbial cells may be protected from destruction in storage, or, more particularly, in the case of those that function at a neutral or alkaline pH, from destruction by the acid environment of the stomach. As enzymes possess pH optima for activity, it is essential that the optimum for a particular enzyme coincides with that in the gut location of its anticipated activity. Adult horses are unlikely to respond to supplementation with active enzymes. Morris-Stoker *et al.* (2001) detected no effect of phytase supplementation on mineral availability when it was given to mature geldings, whereas response in the young of several species has been reported.

Under EU legislation and in Schedule 4 of the UK Feeding Stuffs Regulations 2000, enzymes may be used that are not excluded from application of the Additives Directive by Article 22 of that Directive. Note: it has been demonstrated that adding phytase to a phosphorus-deficient diet for turkey poult chicks improves their bone characteristics. The Statutory Statement on the animal feeding stuff product container shall include the following information:

- the names of the active constituents according to their enzymatic activities;
- the International Union of Biochemistry identification number, i.e. EU number;
- the activity units (expressed as activity units per kilogram, or activity units per litre), if these can be measured by an acceptable method;
- an indication of the period during which the activity units will remain present;
- an indication of any significant characteristics of the enzyme arising during manufacture, as specified in the authorisation concerned; and
- the EC registration number.

DIETARY VITAMIN AND MINERAL SUPPLEMENTS

A simple mixture of cereals, a concentrate protein source such as field beans or soya meal, and hay may be adequate in terms of energy and amino acids to meet the daily needs of a horse, but it is likely to be inadequate in certain of the minerals, trace elements and vitamins for optimum performance, particularly in the longer term. Some raw materials are relatively rich sources, especially of water-soluble vitamins, which makes supplementation of these unnecessary, but such supplementation is unlikely to be hazardous. The natural mineral and trace element contents of roughages and of concentrate feedstuffs tend to be variable and to depend on source and quality. Optimum supplementation is therefore difficult to achieve without chemical analysis.

Where supplements are provided, the total daily intake of each nutrient should fall within the limits imposed for each in Chapters 3 and 4. Where supplements are given in addition to a compounded feed, the amounts from both sources should, of course, be summated. The risks from excess are real for the fat-soluble vitamins,

with the exception of α -tocopherol, and for several of the trace elements. Those elements that have notoriously caused problems are selenium and iodine. The reason for this is that the minimum adequate levels and the toxic threshold concentrations are relatively close and some natural feeds are rich sources. Moreover, for iodine the clinical signs of excess are similar to those of deficiency (see Chapters 3 and 12 for diagnostic methods). Excessive consumption of the major minerals is rarely lethal, but inappropriate rates of intake can certainly contribute to poor performance.

Vitamin-like substances, metabolic enhancers and aids to GI health

Many supplements contain items that are not strictly nutrients, but that have some physiological value to the horse. Care should be taken, where it is essential, that none of these contravenes the rules of competition and racing.

L-carnitine (β -hydroxy- γ -trimethylaminobutyric acid)

A conditionally essential nutrient, L-carnitine is present in substantial amounts in diets composed of animal products, but scarce in feeds derived from plants, and so scarce in horse feeds. It is synthesized from lysine and methionine. Carnitine facilitates the transport of long-chain fatty acids across inner mitochondrial membranes and it may regulate acetyl-CoA:CoA by buffering excess acetyl units during intense exercise. Supplements of 10g L-carnitine given twice daily for two months have doubled plasma carnitine concentration of TBs, but there was neither increased content in nor loss of total carnitine from middle gluteal muscle, associated with intense exercise. However, a supplement of this amount given to broodmares increased the carnitine content of their plasma and that of their foals (Benamou & Harris 1993). The young foal, like the neonatal human, may have a reduced capacity for carnitine biosynthesis, accounting for normal plasma concentrations of about a third those in adults. A few human subjects with leg claudication and angina have demonstrated an improvement in exercise tolerance following carnitine supplementation. The effect of dietary fat supplementation on carnitine need has not been examined, but it is doubtful whether the function of carnitine can be enhanced, except in rare individuals that have low biosynthetic ability. Healthy adult horses probably synthesize adequate amounts from lysine and S-adenosyl-methionine.

Carnosine (β -alanyl-L-histidine)

Carnosine is found in high concentrations in equine skeletal and cardiac muscle and is a major physiochemical buffer. The carnosine content of muscle is positively correlated with the proportion of fast-twitch glycolytic fibres. It is a dipeptide of β -alanine and L-histidine. However, Kavazis *et al.* (2003) were unable to demonstrate any reduction in the rate of skeletal-muscle fatigue of rats, resulting from dietary supplementation with β -alanine and L-histidine.

Glucose

The horse has the ability to store large quantities of glycogen in muscle. Concentrations in well-trained, well-fed horses are 600–700 mmol glucosyl units/kg muscle DM. Attempts have been made to accelerate glycogen repletion rates after extended exercise by use of i.v., or oral, glucose and glucose polymers, but without effect.

Creatine

Creatine was, perhaps, the most widely used supplement in human sport in 1999, as it can increase muscle creatine phosphate and improve performance for short-term high-intensity exercise (Maughan 1999). It had been proposed that supplementary creatine may increase water retention and plasma volume of horses, but Schuback *et al.* (2000) reported that 25 g creatine monohydrate given twice daily had no effect on maximal treadmill performance of Standardbred trotters, as expressed by muscle metabolic activity and total blood volume.

N,N-dimethylglycine

N,N-dimethylglycine (DMG) is an intermediate compound in the metabolism of betaine to sarcosine. In the process there is methyl group transfer to homocysteine with methionine formation. There is evidence that DMG supplementation may delay the onset of fatigue induced by lactic acid. Mature, conditioned, exercised horses supplemented with 1.6 mg DMG/kg BW failed to show typical increases in blood lactic acid concentration (see Chapter 9). Further evidence with trimethylglycine has also shown that post-exercise lactate oxidation is accelerated in untrained horses, but not in trained horses. However, there is a need for more corroborative evidence.

Dimethylsulphone (DMSO₂), methyl sulphonyl methane (MSM)

Dimethylsulphone, methyl sulphonyl methane (DMSO₂, MSM), is included in several horse-feed supplements. It is a naturally occurring sulphur (S) compound in both flora and fauna, which has been shown to provide biologically available sulphur in the metabolism of S-amino acids and it may be an important component of the natural sulphur cycle. MSM is the oxidation product of several natural and synthetic compounds and is an important odoriferous compound in milk. It has the formula: 2(CH₃)SO₂. In laboratory animals with spontaneous chronic arthritis, MSM has been demonstrated to lessen destructive changes in joints when given in relatively large, but quite safe, doses. (For a discussion of glucosamine, methyl sulphonyl methane (MSM) and chondroitin sulphate supplementation see Chapter 8.)

Hesperidine

A very large group of compounds (several thousand), other than carotenoids, exists in plants with red, blue or yellow pigments. They are referred to as *bioflavonoids*, and are polyphenolic. The flavonoids include catechins, proanthocyanidins (condensed tannins) and flavanones (reduced flavonones). Undoubtedly much will be learnt about the fascinating significance of some of these compounds during the next decade. Some potentiate the antiscorbutic activity of ascorbic acid. Hesperidine is an aglycone flavonoid that has been reported to reduce capillary fragility and/or permeability. It may 'spare' vitamin C, possibly by chelating divalent metal cations (Cu^{++} , Fe^{++}), thus serving an antioxidant function. However, the function is speculative, and whether, for example, hesperidine would reduce the frequency of post-race bleeding has not, to the author's knowledge, been tested.

Echinacea angustifolia

O'Neill *et al.* (2002) gave healthy horses a powdered root extract of echinacoside from *Echinacea angustifolia* (Compositae, common name, Coneflower, found in eastern United States), 1.0 g twice daily on their feed. This increased the phagocytic ability of neutrophils, stimulated their migration from peripheral circulation and boosted peripheral lymphocyte counts. It also increased the size (NB: pathological increase in mean corpuscular volume, MCV, occurs in human alcohol toxicity) and concentration of peripheral red cells, haemoglobin concentration and PCV. The authors concluded that *Echinacea* stimulated immunocompetence and acts as a haematinic agent.

Amino acids and health

There has been recent interest in the oral supplementation of both patients following trauma and exhausted long-distance athletes, with branched-chain amino acids and with arginine or glutamine, to overcome leukopenia and immunodepression associated with stress. Glutamine is an important fuel for some cells of the immune system and supplementation may enhance cell-mediated immunity and mucosal immunity by modulation of the inflammatory response and preservation of respiratory and intestinal IgA levels. Although there is some evidence in man to indicate that glutamine could play a role in immunodepression related to trauma and burns, it may not play a mechanistic role in exercise-induced immunodepression. Recently it has been shown that arginine and glutamine consumption increase radical oxygen species production by neutrophils in stressed rats via NO⁻ and polyamine generation (Moinard *et al.* 2003). The mechanism by which they enhance immunity may be related to this stimulation of oxidative metabolism. No equine evidence is presently available (see also 'Ammonia and the alanine vehicle' and 'Protein assimilation', Chapter 9).

Fuller's earth (sodium montmorillonite, sodium bentonite)

Fuller's earth, or bentonite, is a fine greyish-white powder that consists mainly of montmorillonite, a native hydrated aluminium silicate, with which finely divided calcite (calcium carbonate), magnesium and iron may be associated. Fuller's earth is an adsorbent that can take up gases produced in the GI tract. It adsorbs water, swelling to about twelve times the volume of the dry powder. A mixture of 20 g/l water has a pH of 9–10 and it has some buffering capacity in the GI tract, reducing a rapid decrease in pH during starch overload (see Chapter 11). It can also act as a pellet binder. Bentonite is normally available in the feed industry as the sodium salt.

Ergonomic aids

Sodium bicarbonate

(See also Chapter 9.) Lactic acid accumulates in skeletal muscles and body fluids during and following high-intensity exercise. At the pH of normal muscle tissue the lactic acid is highly dissociated into lactate and H^+ ions. These ions accumulate, lowering the pH and reducing the activity of glycolytic enzymes, probably impairing the contraction process of working muscles, expressed as fatigue. H^+ ions are buffered by the bicarbonate buffering system:



and the CO_2 is exhaled, disposing of the H^+ ions. Supplementation of horses with sodium bicarbonate at the rate of about 0.4 g/kg BW in 1 l water approximately three hours before a race of more than two min duration (<1500 m) has been shown to raise venous blood pH and lactate, indicating an improved buffering capacity during metabolic acidosis.

FEED STORAGE

Some organic nutrients and non-nutrients in forages, cereals and compounded feeds deteriorate during storage. Labile, readily oxidized pigments, unsaturated fats and fat-soluble substances are destroyed at differing rates depending on their degree of protection, the environmental conditions, their propensity to oxidation and the presence or absence of accelerating substances. The immediate effects include a reduction in the acceptability of feed to the horse, which is perhaps one of the most discerning and perspicacious of domestic animals over its feed selection.

All the fat-soluble vitamins present naturally in feed – that is, vitamins A, D, E and K – are subject to oxidation, together with the unsaturated and polyunsaturated fatty acids. Rancidity of the latter depresses acceptance, although some stability is imparted by natural and permitted synthetic antioxidants, which are respectively present in, or are used in, mixed feed. Added synthetic sources of vitamins A and E are much more stable than are their natural counterparts, but they contribute very little antioxidant activity. The critical water-soluble B vitamins are fairly resistant to

destruction during normal storage, although riboflavin in feed will be lost where it is exposed to light. Advice given on labels attached to proprietary supplements and feeds should be followed.

Several factors are essential attributes of good feed stores and grain silos. These are a low and uniform ambient temperature, low humidity, good ventilation, absence of direct sunlight, and freedom from rodent, bird, insect and mite infestation. These characteristics imply that feed stores and grain stores should be insulated and windowless, but should be well ventilated and both clean and cleanable. Construction materials should be rodent-proof, stacked feed should be raised from the floor and accessible from all sides, and roofs should be free from leaks. Galvanized bins are generally preferred to plastic bins, which can be gnawed by rodents, but metal bins may be more subject to moisture condensation on the inner surface if the feeds they contain have excessive moisture contents (a maximum of 120 g moisture/kg feed should be achieved). Thus, the choice of store should rest on the level of general tidiness, whether rat infestation is likely, whether all sides of proposed plastic bins can be reached and whether a uniform temperature can be maintained over 24 hours. Uniformity in the latter reduces the risk of temperatures reaching the dew point during its fluctuation.

An Irish study showed that 14% of both Irish and Canadian oat samples were badly contaminated with fungi, although the Irish samples contained slightly more moisture (MacCarthy *et al.* 1976). These fungi grow during the maturation of the crop in the field and normally have a minor role in feed stability. However, high concentrations can affect acceptability to the horse and fungal invasion does detract from the stability of cereals during storage. *Fusarium* species and a few others may produce toxins that subsequently affect fertility or other aspects of animal health.

Storage fungi are another matter; these species can grow in environments with relatively low moisture and high osmotic pressure, known technically as conditions of low water activity. Even in this environment, heating, mustiness, caking, lack of acceptability and eventually the decay of stored grains, oilseeds and mixed feeds can occur, feed stability and nutritional value decline, and fungal toxins may be produced. Species of *Aspergillus* and *Penicillium* are the main culprits and all stored feed, where there are moisture contents that give water activities of 0.73–0.78 at temperatures of 5–40°C, may be invaded by *A. glaucus*. However, *Chrysosporium inops* will spread at moisture contents as low as 150–160 g/kg. Most storage fungi have minimum temperatures for growth of 0–5°C, grow optimally at 25–30°C and do not grow at temperatures above 40–45°C. However, *A. candidus* and *A. flavus* (producing aflatoxin) grow vigorously at 50–55°C and *Penicillium* grows slowly at temperatures down to –2°C.

Uninsulated bins and waterproof sacks subject to variations in environmental temperature are particularly prone to moisture condensation on the inner surfaces, even when the average moisture content of the product is low. Of course, the probability of this occurring increases with greater average moisture levels. Once mould growth is initiated, this generates metabolic water and a vicious circle is established.

Insects, for example grain weevils, beetles and flour mites, not only accelerate deterioration of feed and grain, but also generate both heat and metabolic water, and are vectors of fungal spores. Dirty, badly stored grain will be ripe for the hatching of eggs and the multiplication of these insects and mites. Mites will multiply at moisture levels as low as 125 g/kg and a temperature of 4°C. Insects require slightly higher combinations of temperature and moisture. Cleaning and fumigation of long-stored feed and feed stores are, therefore, desirable. Hygiene and the storage of feed and grain at low temperatures and in a dry condition, without pockets of high moisture, are the greatest assurance for the maintenance of feed quality in the long term.

Weevils and beetles can be seen with the naked eye. Mites are extremely small but their presence can be detected in meals by observation for a minute, during which time movement of fine feed particles should be apparent. There is a characteristic sour smell from mite infestation, whereas with moulding, discoloration of the grain, dust and a fungal smell can be readily detected.

Rodent infestation not only causes a loss of feed, but also both rat and mouse droppings introduce to horses the risk of enteric disease, principally salmonellosis, but also other pathogens, including Tyzzer's bacillus (*Clostridium piliformis*). This bacterium is carried in the GI tract of many wild species, e.g. rodents and lagomorphs (see Chapter 11).

Mould inhibitors, such as calcium propionate, propionic acid, sorbic acid and hydroxyquinoline, have been recommended, but they are really effective only for coating grain and are relatively ineffectual when included in mixed feed. Propionic acid is more effective than the calcium salt.

NATURAL AND CONTAMINANT TOXICANTS IN FEEDS

(See also Chapter 10.) Feed is sometimes naively considered to be a parcel of nutrients, both essential and nonessential. A consideration closer to the truth accepts that natural feeds also contain materials thought either to be inert or to influence the metabolism of other dietary constituents, and substances with nutritional value but which may be present in toxic concentrations. Many natural feedstuffs also contain substances in toxic concentrations with no known nutritive value. Many of these potentially hazardous substances are produced naturally, either by the plants themselves or by organisms infecting them and their products. Finally, there are contaminants that result from human intervention and activity. Table 5.10 gives an arbitrary classification to indicate the extent of the problem, yet the groupings and distinctions drawn are by no means absolute.

Toxicants produced by plants

Those toxicants likely to be consumed by browsing horses will be discussed in Chapter 10. Here our concern is with substances present in seeds used as feeds.

Table 5.10 Classification of naturally occurring toxicants and toxic contaminants of horse feed.

-
- (1) *Naturally occurring toxicants*
 - (a) Protein or amino acid derivatives
 - Lectin (haemagglutinins)
 - Trypsin inhibitors
 - Lathyrogens
 - Nitrates/nitrites
 - (b) Glycosides
 - Goitrogens
 - Cyanogens
 - (c) Miscellaneous
 - Tannins (polyphenols), saponins
 - Gossypol
 - Phytin, oxalic acid
 - Several antivitamin factors
 - (d) Poisonous shrubs and weeds (principally alkaloids)
 - (2) *Moulds and pathogenic bacteria developing owing to bad harvesting, handling and storage*
 - (a) Spores causing respiratory allergies
 - Aspergillus fumigatus*
 - Micropolyspora faeni*
 - (b) Mycotoxins produced by moulds
 - Hepatotoxins, fumonisins
 - Hormonal
 - Other
 - (c) Pathogenic bacteria, or their toxins
 - Pathogenic *Salmonella* spp.
 - Clostridium botulinum* toxin
 - (d) Endophytic fungi in grasses
 - Acremonium coenophialum*
 - A. lolii*
 - (3) *Dietary allergens absorbed from intestines*
 - (4) *Dietary contamination during manufacture*
 - Toxic antibiotics
 - Dopes
 - Pesticide and herbicide residues
 - Nutrients with narrow margin of safety
 - Heavy metals unlikely to have nutritional value
 - Breakdown products of feed constituents
-

Condensed tannins

Condensed tannins comprise a diverse group of water-soluble polyphenolic compounds. They are contained in sorghum grains, lentils and many other legume seeds. Peas and field beans, for example, tend to contain significant amounts of condensed tannins, especially where the seed coat is black (not always a reliable guide) or where field beans are coloured-flowering. They are not toxic but depress appetite and react with, and lower the digestibility of, proteins and carbohydrates in a pH-

dependent manner, if they are present in a sufficient concentration. Autoclaving or pressure-cooking destroys these tannins, but prolonged treatment is required at lower temperatures. The reaction in the intestines occurs with both dietary and endogenous proteins (for example, slightly lowering trypsin activity in the ileum), increasing their faecal loss. The soaking of some beans before cooking may lower digestibility further, as it seems the tannins may diffuse from the seed coat into the kernel where they react with the proteins. If these polyphenols are absorbed, their subsequent detoxification involves methylation. This may lower the protein value of a foal diet, for example, if it is limiting in methionine content.

This enormous group of compounds is under intense scrutiny at present as the spatial configuration of the phenolic hydroxyl groups in some isoflavonoids may confer oestrogenic activity affecting fertility of domestic animals and man. Moreover, the antioxidant activity of the polyphenols in green tea and red grapes is thought to reduce atherosclerotic development.

Lipoxygenase activity

Ground lentils and other ground pulses become rancid rapidly following disruption. The polyunsaturated fatty acids are particularly susceptible to oxidation and the enzyme responsible is lipoxygenase. Heat treatment prior to grinding to destroy the enzyme is an effective preventative measure.

Antiproteases (trypsin, or protease, inhibitors) and lectins

Two widely distributed groups of compounds are known as digestive enzyme inhibitors and lectins (previously known as haemagglutinins). The specific compound, its toxicity and susceptibility to destruction by heat vary among the species of plant within which it is found. Plants producing trypsin inhibitors and lectins include field or horse beans, black grams and kidney, haricot or navy beans. Horse grams, moth bean (*Phaseolus aconitifolius*), certain pulses (also containing amylase inhibitors), groundnuts or peanuts (*Arachis hypogaea*), soya beans and rice germ also contain these substances. Most rice bran fed to horses has had the germ removed, although some residual activity is normally found.

Trypsin inhibitors depress protein digestion, but lectins in commonly consumed legume seeds are considered to be more harmful because they disrupt the brush borders of the small intestinal villi, hamper absorption of nutrients, but apparently allow the absorption of certain toxic substances. They stimulate hypertrophy and hyperplasia of the pancreas and, from evidence of McGuinness *et al.* (1980), can eventually cause adenomatous nodules and pancreatic cancer of exocrine glands. At high concentration they induce rapid depletion of muscle lipid and glycogen in laboratory animals. Tissue catabolism and urinary nitrogen are increased, and thus growth in young stock can be depressed.

The activity of both these groups of substance is destroyed by steam-heat treatment. For example, the trypsin inhibitor activity of field beans is reduced by 80–85%

during steam heating at 100°C for 2 min and by about 90% during treatment for 5 min. However, the trypsin inhibitor and lectin activities of kidney beans are very stable because treatment for two hours at 93°C is necessary for adequate destruction. Kidney beans are therefore generally unsuitable for non-industrial processing and should not normally be fed to horses.

Lathyrism caused by β (γ -L-glutamyl) aminopropionitrile and L- α , γ -diaminobutyric acid

β (γ -L-glutamyl) aminopropionitrile and L- α , γ -diaminobutyric acid are present in various species of *Lathyrus*. The horse is particularly subject to intoxication. The Indian or grass pea, after long periods of feeding, causes a condition known as lathyrism, which is exemplified in the horse as a sudden and transient paralysis of the larynx with near suffocation brought on by exercise. This is associated with a degenerative change in the nerves and muscles of the region and profound inflammation of the liver and spleen. Other closely related species, including sweet pea (*L. odoratus*), wild winter pea (*L. hirsutus*), singletary pea (*L. pusillus*) and everlasting pea (*L. sylvestris*), can also cause lathyrism. Although the whole plant contains the toxin, the seeds appear to be the most potent source and it is only partially destroyed by heat.

Goitrogens

Goitrogenic activity is caused by goitrins, which are derived from glucosinolates found in many members of the Cruciferae family, including cabbages, rape and mustard. Goitrins are released by enzymes contained within the plant and the destruction of these enzymes by heat treatment to a large extent eliminates the potential hazard. The effect of goitrins is not counteracted by additional dietary iodine, but further enzymatic metabolism of certain goitrins can release isothiocyanates and thiocyanates. The antithyroid effect of these substances on young horses, in particular, can be overcome by dietary iodine (see Chapter 3). The enzymes are destroyed by adequate heat treatment. The slight antithyroid effect of uncooked soya beans is said to be overcome by additional iodine.

Cyanogenic glycosides and hydrocyanic acid

Cyanogenic glycosides release hydrocyanic acid (HCN) on soaking the seed. Feeds containing these HCN glycosides should be given in small amounts and dry. Larger amounts require preheating to a sufficient temperature (adding to boiling water) to destroy the enzyme that releases the HCN. The glucoside in *Phaseolus lunatus* remains stable to cooking. Vicine is the pyrimidine glucoside in *Vicia faba* responsible for the haemolytic anaemia of favism in subjects deficient in glucose-6-phosphate dehydrogenase but favism is generally unrecognized in horses. Glycosides, derivatives of α -hydroxynitriles, present in lima beans, sorghum leaves,

linseed and cassava (tapioca) (*Manihot esculenta*), generate HCN when acted on by the specific enzymes the plants contain. HCN can cause respiratory failure by inhibiting cytochrome c oxidase (EC 1.9.3.1). Again, as the poison is released by enzyme activity, heat treatment will ensure safety, so long as prolonged storage of, for example, moist seeds or cassava roots has not led to some accumulation of HCN. The suppression of enzymatic activity is another reason for the importance of dry storage of certain uncooked feedstuffs. HCN can also react with any thiosulphate present, producing thiocyanate, which is itself responsible for thyroid enlargement after prolonged feeding.

Gossypol

Gossypol, occurring in many strains of cottonseed, is the reason the meal is not widely used in horse feeds. In both its bound and free forms, the pigment gossypol reacts, incompletely, with cottonseed protein to depress appetite and protein digestibility and therefore to reduce the efficiency of amino acid utilization; but its toxicity can result in death caused apparently by circulatory failure. The pigment also reacts with dietary iron, precipitating it within the intestines. Fairly large additions of supplementary iron to the diet will then promote further precipitation, which partially suppresses the adverse effects of gossypol.

Antivitamins

Several antivitamin factors are present in animal and vegetable feeds, but most are of little significance to horses. A thiaminase present in the bracken fern (*Pteridium aquilinum*) is a cause of bracken poisoning, which is counteracted with large doses of thiamin, and the antivitamin E factor present in raw kidney beans is partly destroyed by cooking. Sulphonamides have an antimicrobial action and depress intestinal vitamin K synthesis.

Phytins and oxalates

High levels of phytic acid, or its salts, present in many vegetable seed products, when consumed in large quantities will interfere with the availability of calcium and several trace metals, particularly zinc. Oxalates detectable in many plants and present in high concentrations in certain tropical species of grass have been reported to kill cattle and cause lameness in horses, owing to precipitation of calcium (see Chapter 10).

Nitrates

Green fodder containing more than 5 g nitrate/kg DM fed alone may cause digestive disturbances and respiratory and circulatory abnormalities. The rapid growth of pasture after high rainfall and excessive use of nitrogen fertilizers can lead to high

concentrations of nitrates in the herbage, and contamination of water supplies through the leaching of soils. Although nitrates are only slightly toxic, they can be reduced to nitrites before or after consumption. High levels of nitrites may accumulate in plants after herbicide treatment and during the making of oat hay, owing to nitrate reduction encouraged by inclement weather. In the body, nitrites convert blood haemoglobin to methaemoglobin, which is unable to act as an oxygen carrier. Large intakes therefore cause death. Pigs are probably more susceptible than are horses, which appear to react similarly to ruminants.

Alkaloids

In South Africa, America and Australia numerous species of the legume *Crotalaria* (one species of which causes Kimberley horse disease) have proved very poisonous in horses. Lesions are induced in the liver by pyrrolizidine alkaloids similar to those found in ragwort (*Senecio*). Alkaloids are considered in Chapter 10 under Grazing management. Other details are found in Table 10.17. Green potatoes (*Solanum tuberosum*) contain the alkaloid solanine. Horses are killed by eating quantities of potatoes that would not affect ruminants, even when the tubers are not apparently green.

Moulds

The effects of moulds are of two types:

- (1) Certain mould spores in large numbers in badly harvested and stored roughages and in cereals can cause a respiratory reaction (allergy) when inhaled by sensitive horses (see Chapter 11).
- (2) Many mould species in the appropriate conditions of temperature and humidity produce toxins which have a variety of metabolic effects.

Mycotoxicosis

Fumonisin

Fusarium moniliforme (mating type A, of *F. moniliforme*), or *F. proliferatum*, generally growing before harvest on maize, produce water-soluble fumonisins, and possibly other toxins. These toxins cause damage to the equine liver, which is found on pathological examination to be small, firm and yellow, with centrilobular necrosis and fibrosis (Thiel *et al.* 1992). The disease in horses, donkeys and mules is also specifically characterized by liquefactive necrotic lesions in the white matter of the cerebral hemispheres (equine leukoencephalomalacia, LEM), causing blind staggers, whereas in man oesophageal cancer occurs (Bryden 1995, 1998). The mating type of *F. moniliforme* found on sorghum does not produce fumonisins (Bryden 1998, personal communication). Fumonisin bear a structural relationship to the

sphingoid base, sphingosine, and so interrupt sphingolipid metabolism in the central nervous system with a rise in serum sphinganine. Of six fumonisins, FB_{1a} and FB₂ are thought to be the only significant equine toxins. The time taken to develop clinical signs depends on dose, but it may be within eight days and include apathy, somnolent appearance with protruding tongue, reluctance to move backwards, aimless circling and ataxia (Nelson *et al.* 1993).

F. moniliforme is an aggressive colonizer of maize pre- or post-maturity (Bryden *et al.* 1998) and especially that infested by weevils (Smalley 1992). FB₁ is the major phytotoxin (Lamprecht *et al.* 1994), but not acutely so (Gilchrist *et al.* 1992). The fungus can colonize, reproduce and produce toxins on a wide range of mature plant products, plant debris and on autoclaved maize seed when incubated at 20–22°C (Marijanovic *et al.* 1991; Nelson *et al.* 1993), or when grown on autoclaved rice for 28 days at 22–26°C (Mirocha *et al.* 1992). It is a facultative parasite (Gilchrist *et al.* 1995) and may be described as an endophyte (Bryden, personal communication; Marasas *et al.* 1988a). Of 675 visually normal maize kernels, 60% were infected with *F. moniliforme* and only seven samples were negative for FB₁ out of 100 samples of normal Australian maize for human and animal food (Bryden *et al.* 1998). Almost half these animal feed samples contained FB levels of 5 mg/kg and above (mean 7.8 SE 1.01 mg/kg). The highest concentration recorded by Bryden, 40.6 mg/kg, was detected in maize used for equine feed, causing LEM, and 84% of Australian samples drawn from both field and storage were found to be infected with *F. moniliforme* (Pitt & Hocking 1996). Fumonisin B₁ has world-wide distribution. The range of content in maize grain samples from Iowa was 2.5–3.5 mg/kg and in South Africa 0.4–1.8 mg/kg.

Fumonisin B₁ and B₂ feed concentrations of as little as 1.3 and 0.1 mg/kg of feed, respectively, have been associated with LEM (Thiel *et al.* 1992) and fumonisin intakes of as little as 0.6 mg/kg BW/day (approximately 20 mg/kg air-dry feed) caused pathological effects in horses (Thiel *et al.* 1992), although most cases of LEM have been associated with concentrations of >500 mg/kg. A daily intravenous dose for seven days of 0.125 mg fumonisin B₁ per kg BW caused neurotoxic signs in a horse on day eight and oral doses of 1.25–4.0 mg FB₁ per kg BW (approximately equivalent to 40–130 mg/kg feed) caused pathological effects in horses (Kellerman *et al.* 1990). At autopsy the lesions included oedema of the brain and bilaterally symmetrical focal necrosis in the medulla oblongata (Marasas *et al.* 1988b).

Colic and mycotoxins

Several mycotoxins have been examined for their propensity to cause colic. At the concentrations found in contaminated feedstuffs only T₂ toxin, at concentrations >50 µg/kg feed, and zearalenone, at concentrations >70 µg/kg feed, have caused colic in horses. The author has witnessed several cases of impaction colic and death amongst Arabian horses consuming slightly mouldy timothy hay that was otherwise of good quality. No colic has been observed with aflatoxin, deoxynivalenol or with fumonisin B₁.

Ergotamine, ergometrine

There are numerous historical references to the consequences of consuming ergot of rye, namely abortion and other effects of blood-vessel constriction. The mould concerned, *Claviceps purpurea*, also infects rye grasses and some other grasses, and so in high concentrations can be a hazard in pasture and hay.

Aflatoxin

Several reports, especially from Thailand and the USA, have recorded deaths or brain, heart and particularly liver lesions, and haemolytic enteritis in horses receiving aflatoxin produced by *Aspergillus flavus* at levels of less than 1 mg/kg contaminated cereals, peanuts and even hay. Horses and ponies seem to be more susceptible to acute aflatoxicosis than are pigs, sheep and calves, as daily intakes of 0.075 and 0.15 mg aflatoxin B₁/kg BW are lethal for ponies in 36–39 days and 25–32 days, respectively (Cysewski *et al.* 1982).

Zearalenone

The toxin zearalenone, produced by *Fusarium* species, causes vulvovaginitis and reproductive failure in females of several domestic species. Whereas aflatoxin usually develops during storage, this toxin may develop pre-harvest. Although no reports of effects in horses are known [cf. 'Equine dysautonomia (EGS, grass sickness)', Chapter 10], it could well cause breeding irregularity in them. Many other fungal toxins with a wide variety of effects and significance also exist (also see 'Zearalenone', Chapter 10).

Dietary allergens (other than mould spores)

Dietary allergens are not contaminants, but certain horses can react to normal protein constituents of feed (Plate 5.4). The effects, which include respiratory and skin lesions, are normally overcome, according to the author's experience, by removal of the offending source from the diet. Problems of cross-reactivity in which related sources of proteins yield similar reactions can, however, pose problems of interpretation.

Human intestinal epithelial cells take up and process food antigens (Heyman 2001). Among the peptidic metabolites generated during transepithelial transport of luminal antigens, some have a molecular mass compatible with a binding to restriction (major histocompatibility complex) molecules. These can be up-regulated on enterocytes, especially in inflammatory conditions, such as gastroenteritis. Nutritional factors can influence the properties of this epithelial barrier and immune response to luminal antigens. In particular, Sanderson (2001) indicated that changes in diet can alter the expression of genes in the gut epithelium encoding for proteins that signal to the mucosal immune system. The epithelium thus acts as a transducing



Plate 5.4 A two-year-old TB gelding with widespread 'bumps' on the head, neck, shoulders, ribs and flanks. An allergic reaction to bran and oats was detected in the blood serum. The horse recovered over several months when its diet consisted of a high-fibre cubed diet, low in cereal and with water *ad libitum*.

Table 5.11 Approximate maximum tolerable dietary concentrations of heavy metals.

	mg/kg feed DM
Arsenic (As)	2
Cadmium (Cd)	10
Fluorine (F)	50
Iodine (I)	1
Lead (Pb)	20
Mercury (Hg)	0.2
Molybdenum (Mo)	200 (excluding any possible interaction with Cu availability)
Selenium (Se)	2
Aluminium	950

monolayer signalling between the contents of the intestine and the mucosal immune system.

Heavy metal and mineral contamination from pastures

(See also Frape 1996, 2002b) The approximate maximum tolerable dietary concentrations of heavy metals and some other elements (these concentrations are influenced by the concentrations of essential minerals and trace elements) are shown in Table 5.11.

Dietary aluminium in excess of 1500 mg/kg causes a reduction in P absorption and an increased P requirement in ruminants and horses, whereas, over four weeks, diets containing 930 mg Al/kg had a negligible effect on nutrient digestibility and mineral metabolism in adult TBs (Roose *et al.* 2001).

Lead is one of the commonest causes of poisoning in cattle, sheep and horses. Signs of toxicity are more frequent in young horses and include lack of appetite, muscular stiffness and weakness, diarrhoea and, in an acute form, pharyngeal paralysis and regurgitation of food and water. Lead accumulates in the bones and as little as 80 mg/kg diet may eventually cause toxic signs, which are sometimes precipitated by other stresses. Natural feeds with 1–5 mg lead/kg cause no problems. The acute lethal dose is 1–1.8 g/kg BW as lead acetate or carbonate. The chronic lethal dose depends on many factors but is said on average to be about 12 mg of lead/kg BW daily for 300 days.

The contamination of pasture with lead, cadmium and arsenic – derived from mine workings, dumping or sewage sludge, aerial dust and water erosion, even from car batteries and lead shot – are local risks. Where the pasture is dense, undoubtedly the greater problem arises from surface contamination of the plants, but where it is sparse, soil, either rich in these heavy metals or contaminated by them, can be consumed in sufficient amounts to cause problems. Lead shot is somewhat hazardous to horses, but, because they close graze, consumption can be greater than might be generally appreciated, followed by some solution in the stomach. Where grass is

ensiled, entrapped shot partly dissolves during fermentation and highly toxic levels of 3800 mg soluble lead/kg DM have been detected in England by the author (Frape & Pringle 1984). Whereas lead seems mainly to contaminate the surfaces of plants, cadmium is readily absorbed and is accumulated from soils rich in this element. Of common pasture species, the daisy (*Bellis*) accumulates 60–80 mg cadmium/kg (30 times as much as in grasses) from contaminated soils (Matthews & Thornton 1982).

Pastures to the leeward of steel and brick works may amass abnormally high concentrations of fluorine. The horse is probably less subject to fluorosis than are cattle and sheep, but damage to its bones and teeth has been induced by this element. However, it will tolerate 50 mg/kg feed for extended periods. Mercury poisoning expressed as colic and diarrhoea has arisen in horses as a consequence of the mistaken use of dressed seed as a feed. Chance exposure from other sources is unlikely.

The required trace elements (see also Chapter 3) – iodine, selenium and molybdenum – may be consumed in toxic quantities following natural accumulation in vegetable materials. Seaweed can be a source of excessive iodine, and certain accumulator plants store large quantities of selenium from selenium-rich soils. When these plants die it is said that they in turn deposit selenium in a form readily absorbed by neighbouring plants. Many selenium-rich areas are sparsely covered and the consumption of soil rich in the element is another source of risk. Molybdenum is readily absorbed by most plants from soils containing excessive amounts. The ingestion of soil rich in iron and sulphur is known to reduce copper absorption in grazing animals. Season and the extent of soil drainage can influence the accumulation of several metals in herbage. Concentrations frequently tend to be higher during the winter months (see also Chapters 3 and 10).

Pesticide residues

Many normal feeds contain trace amounts of pesticide residues but, excepting gross contamination through negligence, the amounts normally detected are insufficient to cause any problem to horses (after herbicide treatment, pastures should be rested for two weeks before grazing is permitted). The rodenticide zinc phosphide (now rarely used) has been known to be consumed in lethal amounts by horses when it may release and eject from the stomach poisonous phosphine (PH_3). If this poison is suspected, a nasogastric tube should only be used in the open air. The acute lethal dose in horses is 20–40 mg/kg BW, or about 15 g for a horse (about 5–10 g for a pony).

Ethylene glycol

Swor *et al.* (2002) successfully treated a gelding that had consumed ethylene glycol (EG) and presented with tachycardia and discomfort. Toxicity of EG is attributable to its oxidation products, through the action of alcohol dehydrogenase (EC 1.1.1.1) (ADH). ADH has greater affinity for ethanol than for EG and so by early intravenous administration of a 20% solution of ethanol at 2.5 ml/kg BW the metabolism of

EG is arrested, leading to its renal excretion unaltered. Intermittent infusions were continued for 32 hours to maintain a therapeutic blood level of ethanol, after which the horse was discharged with no adverse sequelae. A recent alternative to ethanol therapy in man is administration of fomepizole, or 4-methylpyrazole (Antizol®) (Brent 2001). Like ethanol, fomepizole inhibits alcohol dehydrogenase. It does so without producing serious adverse effects, although no evidence of its application to equine toxicosis has been published to date.

FEED ADDITIVES

Domestic animal feed-additive drugs

Several drugs are used in the feed of farm animals to promote growth, to counteract diarrhoea and parasitic infection and to influence the carcass. Most of these drugs have little, if any, ill effect on horses when present in the diet at normal feed levels, or when horses are mistakenly given feed containing antibiotics intended for other species. Higher dosages are a different matter. Although framycetin sulphate is sometimes useful in cases of flatulence, or fermentative colic, the persistent use of some antibiotics, especially oxytetracycline, may cause a severe upset to the intestinal flora, possibly including a fungal overgrowth, precipitating acute and intractable diarrhoea, lethargy and lack of appetite. Two other drugs (monensin and lincomycin), monensin in particular, can have severe toxic effects in horses when given at normal feed rates.

Ionophore antibiotics

Ionophores are polyether carboxylic antibiotics given to poultry for the control of coccidiosis and to ruminants to improve feed utilization. There are presently six of these carboxylic ionophores: monensin, lasalocid, salinomycin, narasin, maduramicin and laidlomycin. Whereas the horse is subject to intoxication from each of these at feed levels, there is a dietary threshold concentration below which no adverse effect has been observed. Above this level severe colic, sweating, trembling and occasionally haematuria can occur. Reliable and quantitative equine data, however, are not available for all of these chemicals (Table 5.12).

Monensin sodium

Monensin is fed to beef cattle for promoting growth and to poultry as a coccidiostat. Poultry feed containing 100 mg monensin/kg, the normal feed level, has severe toxic effects when fed to horses. At a level of 30 mg/kg in the feed, horses experience a reduced appetite and uneasiness, although Matsuoka *et al.* (1996) indicate that horses can tolerate the highest usage rate for cattle of 33 mg/kg feed. At a level of 100 mg/kg (about 2.5 mg/kg BW) in a diet fed continuously, it is lethal in a matter of 2–4 days to about half the individuals. Horses present signs, in the author's experi-

Table 5.12 Ionophore antibiotic toxicity.

Antibiotic	Active chemical usage rate (mg/kg feed DM)	Equine lethal oral dose (LD ₅₀) (mg/kg BW) ¹
Salinomycin	60	approx. 0.6
Narasin	70	approx. 0.7
Monensin	100–120	1.38–3 ²
Lasalocid	30–50	21.5

¹ Lethal dose in mice is about ten times higher.

² Matsuoka *et al.* (1996) found an LD₅₀ of 1.38 mg/kg BW for a single dose by gavage using mycelial monensin.

ence, of anorexia, posterior weakness, profuse sweating, tachycardia, occasionally muscular tremors, polyuria, myoglobinuria (dark-brown urine), elevated urinary potassium, elevated serum levels of muscle enzymes, progressive ataxia and recumbency. Post-mortem examination shows myocardial degeneration and monensin can normally be confirmed by analysis of stomach contents. In the early stages of toxicity, recovery can frequently be achieved by removing the offending feed and dosing the horse with mineral oil, although it may suffer permanent heart damage with increased risk during hard physical exertion.

Lincomycin

Lincomycin is an antibacterial drug sometimes included in pig feed. It is less toxic than monensin in horses, but above dose levels of 80 µg/kg BW daily (5 mg/kg total diet fed continuously) metabolic signs of toxicity and evidence of liver damage have been observed by the author.

PROHIBITED SUBSTANCES

The list of proscribed drugs or prohibited substances embraces a very high proportion of the drugs permitted in livestock feeds by EU legislation, and the detection of any of them, or their recognizable metabolites, in urine, blood, saliva or sweat will lead to the disqualification of a horse subject to the Rules of Racing. Thus, apart from drugs that might be used directly in horses, any antibiotic, growth promoter or other drug used for feeding to poultry, pigs or ruminants must not be detected in any of the above fluids. Of the proscribed drugs, most are unlikely to be present in feed. However, some drugs acting on the cardiovascular system, some antibiotics and one or two anabolic agents have been detected in contaminated feed ingredients – oats, soya-bean meal, bran – and in feed additives, or they may be present in feeds for other classes of stock mistakenly fed to horses. In practice, those causing chief concern are the xanthine alkaloids – theobromine, caffeine and its metabolite theophylline.

Caffeine is present in tea, coffee, coffee by-product, cola nuts, cacao and its hull, which is available as a by-product, and in maté leaves. Tea dust contains as much as 1.5–3.5% caffeine, whereas coffee by-product, as normally available, contains only some 200mg caffeine/kg. Small amounts of theophylline are found in tea, but as much as 1.5–3% theobromine is typically present in cacao beans, and its waste-product, the hull, contains as much as 0.7–1.2%. Theobromine also occurs in cola nuts and tea. The widespread international traffic in coffee and cocoa beans, and in their by-products, constitutes a formidable risk through their contamination of the means of transport, from ships at one extreme to hemp sacks at the other. These means, in their turn, put in jeopardy cereals, pulses and other raw materials moved from one place to another, leading to infringements of the Rules of Racing through the inadvertent consumption of contaminated batches of these feeds. Gross contamination and its control in animal feedstuffs has been discussed in a code of practice published by the United Kingdom Agricultural Supply Trade Association (UKASTA) (1984).

After an oral dose with caffeine, about 1% appears unchanged in the urine, the excretion of which is almost complete after three days. About 60% of the caffeine is excreted in the urine as metabolites, including theophylline and theobromine. Traces of the latter may continue to be excreted for up to ten days, whereas theophylline excretion is virtually complete after 4–5 days. Thus, the unintentional use of these alkaloids, or of coffee or cocoa wastes containing them, can result in their detection in the urine for up to ten days, and caffeine is, moreover, demonstrable in urine within one hour of an oral dose. Great care must therefore be exercised in the feeding of race and competition horses during such a period. The extent of the excretion curves in urine of various other drugs has been determined, but certainly not those of all antibiotics similarly excreted.

Both caffeine and theobromine are readily absorbed from the intestinal tract and soon impart their effects of cardiac and respiratory stimulation and of diuresis. However, tests have shown that they affect horse speed only when they are used in high doses. Experiments have shown that theobromine can be detected in the urine of TB geldings when they receive as little as 1 mg theobromine (in the form of cocoa husk)/kg racehorse cubes and 7 kg of those cubes are given daily, divided between two feeds. The threshold level for theobromine in the urine, accepted by the Stewards of the Jockey Club, is 2 µg/ml urine, when HPLC is used in the analysis (Haywood *et al.* 1990).

Three other proscribed compounds occurring naturally in plant tissue are: hyoscyine, present in the Jimson weed (found in soya bean fields); hordeine, present in germinating barley, alfalfa and canary grass (*Phalaris canariensis*); and morphine, present in poppy (*Papaver somniferum*) seeds and seed capsules.

Growth hormone

There is increasing use of exogenous bovine growth hormone in TB studs. This increases the extent of ‘double muscling’ and rate of growth with little effect on

ultimate height. Some of the author's evidence suggests it may also increase the risk of developmental orthopaedic disease.

STUDY QUESTIONS

- (1) How would you set about changing an existing stable over from hay to haylage?
- (2) What factors should be considered when it is proposed to introduce fat into a rationing system?
- (3) A number of feed processing techniques have been introduced during the last two to three decades. Have any of these been of particular value to horse feeds? If so, which and why?
- (4) If probiotics are to be introduced, what checks should be made concerning: (a) the product and (b) the feed system for their use?

FURTHER READING

- Haywood, P.E., Teale, P. & Moss, M.S. (1990) The excretion of theobromine in Thoroughbred race-horses after feeding compounded cubes containing cocoa husk – establishment of a threshold value in horse urine. *Equine Veterinary Journal*, **22**, 244–6.
- McDonald, P., Edwards, R.A. & Greenhalgh, J.F.D. (1981) *Animal Nutrition*. Longman, London and New York.
- Moss, M.S. & Haywood, P.E. (1984) Survey of positive results from race-course antidoping samples received at Racecourse Security Services Laboratories. *Equine Veterinary Journal*, **16**, 39–42.
- United Kingdom Agricultural Supply Trade Association (1984) *Code of Practice for Cross-contamination in Animal Feeding Stuffs Manufacture*. Amended Code, June 1984. UKASTA, London.

Chapter 6

Estimating Nutrient Requirements

What good receipt have you for a horse, that hath taken a surfeit of provender. This comes commonly to such horses as are insatiable feeders and therefore it is requisite that they be dieted, especially if they have too much rest, and too little exercise.

T. De Gray 1639

The formulation of adequate diets requires knowledge of three types of information:

- (1) the requirement of the horse for each of the nutrients and energy, affected principally by the horse's size and function;
- (2) the nutrient composition of each of the appropriate feeds available; and
- (3) the capacity of the horse for feed.

Tables have been derived to provide the information necessary for (1) and (2), and some discussion is appropriate first concerning (3).

RELATIONSHIP OF CAPACITY FOR FEED TO BODY WEIGHT

The daily requirements of horses and ponies have been estimated in terms of the amounts of each nutrient – minerals, trace elements, vitamins and amino acids (or, more realistically, protein) – required per day for the various functions of maintenance, growth, lactation and so on. The normal vehicle for these nutrients is the daily feed, and if a particular horse were to consume twice as much feed as another horse fulfilling the same tasks then it might be reasonable to suppose that the nutrient concentration in the diet of the first horse need be only half that in the diet of the second. Thus, in order to make useful statements about dietary composition and to facilitate calculation of adequate diets, it is necessary to predict reliably the appetite of a horse, or group of horses, for feed, or more specifically, to predict appetite for dry matter. The appetite and capacity of horses for acceptable feed are regulated by five dominant and related factors:

- (1) the volume of different parts of the intestinal tract;
- (2) the rate of passage of the digesta;
- (3) the concentration of certain digestion products in the intestine;

- (4) the energy demands of the horse; and
- (5) the energy density and its chemical form in feed.

(3) seems to control meal size and (2) will be modulated by the physical form of the feed. (1) is controlled by the body size of the animal, but to some extent is modified by breed and adaptation.

The most common, easily corrected deficiencies in home-prepared feed mixtures are those of calcium, phosphorus, protein, salt and possibly vitamin A. However, in addition to water, the fundamental, immediate and long-term need of the horse is for a digestible source of dietary energy. Ideally, in situations of moderate work or productivity, the energy demands should just be met by the appetite and capacity of the horse for feed. Not only capacity, but also energy requirement for a variety of functions, is closely allied to body weight, although this weight varies from day to day according to the amount of gut fill. Therefore, a means of estimating weight is fundamental to any rationing system.

In the absence of facilities for weighing horses, the most reliable predictions include a measurement of the heart girth following respiratory expiration (Fig. 6.1). As conformation changes with age and differs among breeds, the measurement of girth alone is bound to yield only an approximate estimate. Some improvement on this is achieved by inclusion of the length of the horse from the point of the shoulder to the point of the buttocks (Fig. 6.1). If one wishes to include length, then the equation given under 'Measurement of energy expenditure in maintenance and exercise', below, provides the appropriate relationship.

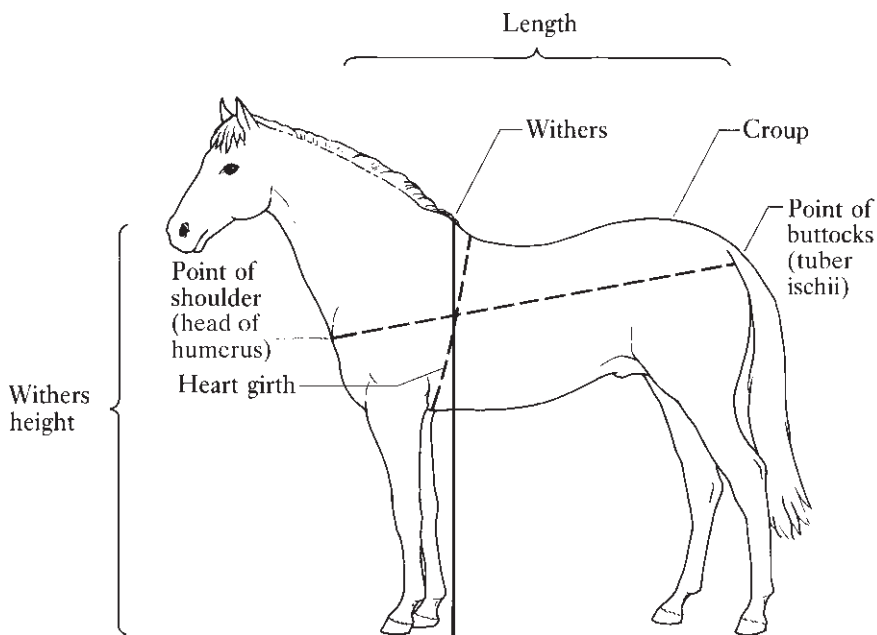


Fig. 6.1 Linear body measurements used in estimating body weight of horses and ponies.

For many, withers height will be a more familiar index of size. Figure 6.2 shows its approximate relationship to body weight for several types of horse and pony, and Fig. 6.3 delineates the change in withers height with age during normal growth. Suggested average daily allowances for horses of different liveweights are indicated in Fig. 6.4. Average capacity values for horses given in Fig. 6.4 are in accord with published values for adult horses. These are approximately 10% higher than the voluntary intake values of 28–29 g/kg BW for a mixed diet in Arabian and

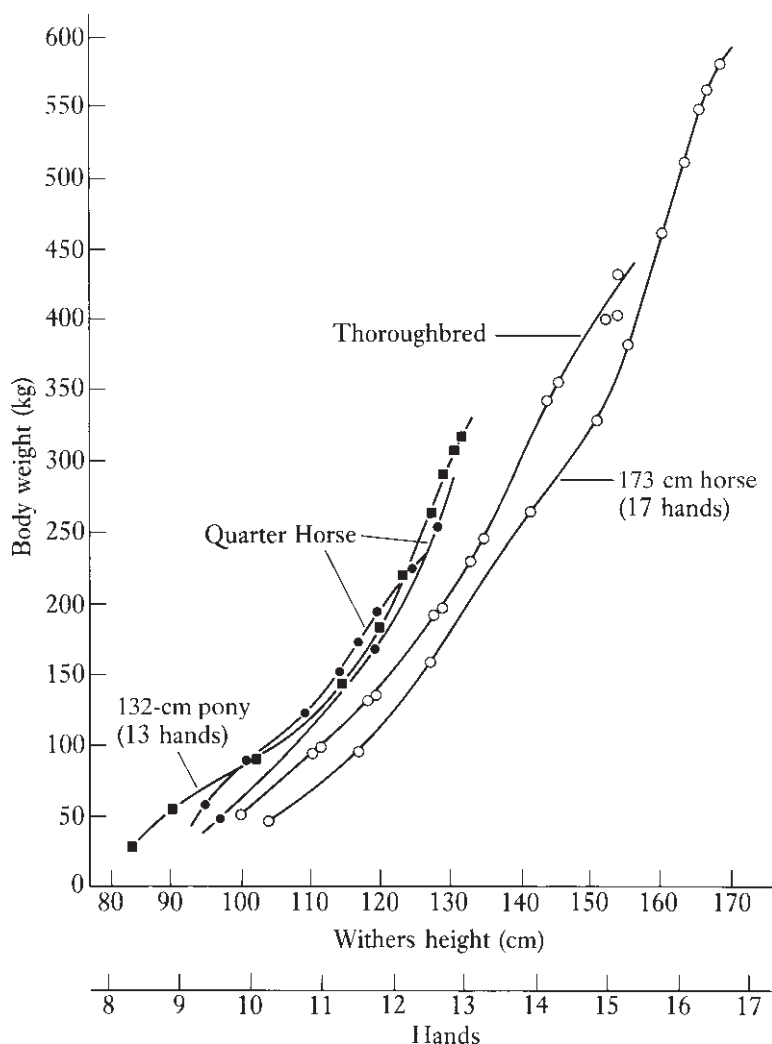


Fig. 6.2 Relationship between body weight and withers height in normally growing horses and ponies (1 hand = 10.16 cm). Note that horses and ponies achieve mature height before mature weight so the curves must be concave upwards as maturity is approached (*upper end of curve*). Furthermore, as the curves are not coincident, withers height is generally not a good predictor of body weight (data from Green 1961, 1969; Hintz 1980a; Knight & Tyznik 1985; R. W. W. Ellis & R. A. Jones 1984, personal communications).

Quarter Horse foals five to eight months of age (Turcott *et al.* 2003). The allowances given to idle horses would be lower than those shown, whereas lactating mares will consume more feed. Furthermore, hard-worked animals, such as TBs in advanced training for racing will be entitled to consume amounts near their capacity, although their appetites may decline when vigorous exercise is practised routinely. Observations in the USA showed that among seven racing stables the average daily intake of concentrate by three to four year olds was 6.16kg (4.9–7.5kg) and that of hay 9.37kg (6.4–11.9kg) (Glade 1983a). Comparable observations among two to four year olds in Newmarket by the author showed that the concentrate intake averaged 8.15kg and that of roughage 5.5kg daily per horse. The lower intake of roughage in the UK may reflect the generally poorer nutritional quality of this feed as supplied to horses. In the American study, the horses were estimated to average 496kg body weight whereas those in the UK were about 480kg.

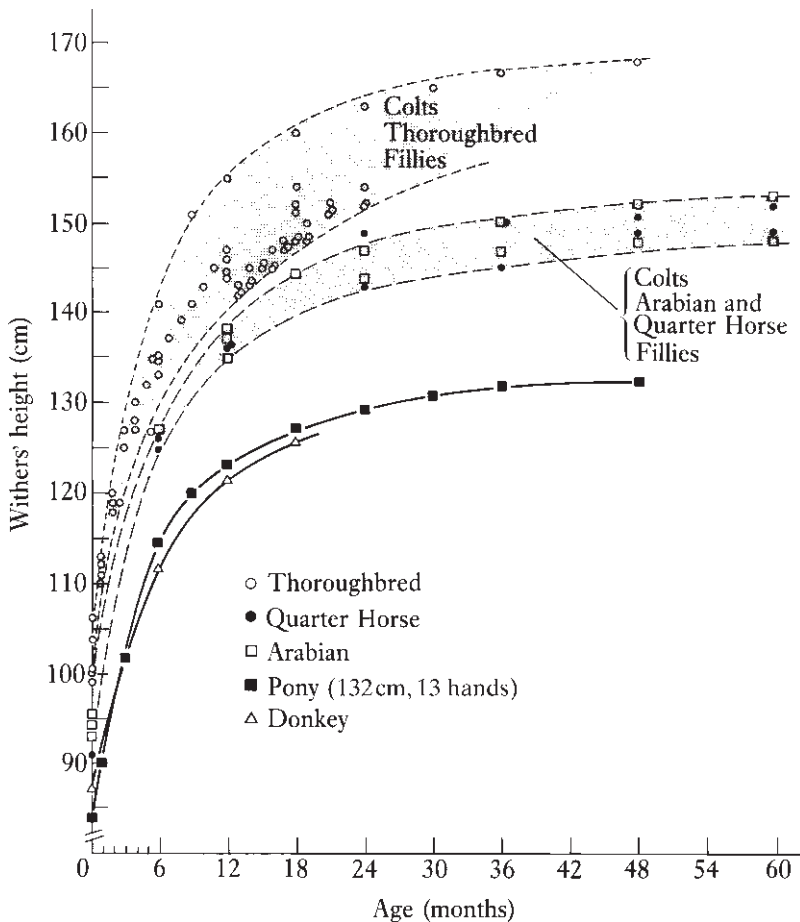


Fig. 6.3 Expected withers height of normally growing horses and ponies at various ages (1 hand = 10.16 cm) (data from Green 1961, 1969; Hintz 1980a; Knight & Tyznik 1985; R. W. W. Ellis & R. A. Jones 1984, personal communications).

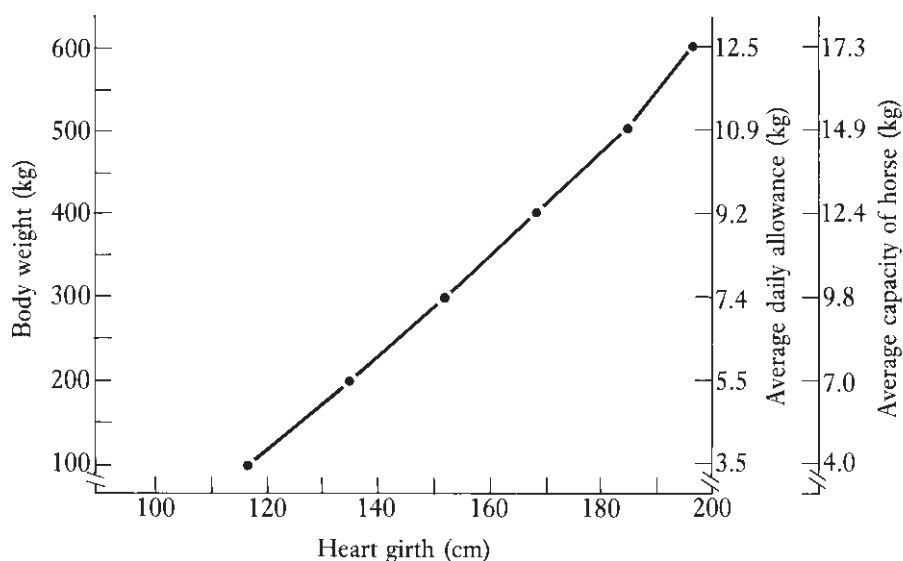


Fig. 6.4 Relationship for horses and ponies between heart girth and average liveweight, average daily capacity for concentrates and forage, and average allowance of concentrates and forage (12% moisture).

Measurement of energy expenditure in maintenance and exercise

Maintenance energy expenditure is eventually expressed as heat evolved at constant body temperature, but the expenditure for production furnishes either work done or useful derivatives, such as growth, milk, products of conception etc. Heat evolved may be measured by *direct calorimetry*, but energy expenditure for maintenance and for work are both more simply measured by *indirect calorimetry*. This method has been widely applied in equine nutrition research and so it is appropriate here to summarise briefly the critical features of the method.

How is the energy requirement of maintenance determined?

The measurement of the inherent activity of the horse's organs and tissues, independent of exercise and other extraneous factors, is a measure under basal conditions and is called the measurement of *basal metabolic rate* (BMR). During these conditions the body is at absolute rest. The specific dynamic action of digestive processes is absent and air temperature and air movement are such that sweating is minimal and the sympathetic nervous system is not activated to help maintain body temperature. For practical purposes it is more appropriate to measure maintenance energy expenditure when feeding is adequate to allow the maintenance of body weight, when movement is that of standing and walking slowly without a rider.

These maintenance requirements are a function of body weight. This must be obtained, either by weighing the horse, or by estimation from linear measurements, as follows (Carroll & Huntington 1988):

$$\text{Body weight (kg)} = \frac{\text{heart girth (cm)}^2 \times \text{length (cm)}}{11\,877}$$

(Carroll & Huntington 1988)

Carroll & Huntington examined the effect of the condition score (maximum 5) on the accuracy of weight prediction. They found a significant difference in the best constant (denominator value) of the equation above between animals with a condition score of:

2.5 or less, for which the denominator is 12 265 cm³/kg,

and those for which the score is:

≥ 3 , for which the denominator is 11 706 cm³/kg.

These denominator values may be used to replace '11 877' to improve the estimate of body weight, if the condition score has been determined. Age affects conformation and so also the preferred denominator. Wilson *et al.* (2003) derived a constant of 10 080 cm for weanling horses. Staniar *et al.* (2003), with a carefully conducted study of TBs from birth to 17 months, fitted a slightly cumbersome broken-line model with different coefficients. This derived body volumes V_{t+1} from girth (G), carpus circumference (C), length of front leg (F) and body length (B):

$$V_{t+1} = [(G^2B) + (C^2F)]/4\pi$$

Where $V_{t+1} < 0.27 \text{ m}^3$, BW (kg) = 1093 V_{t+1} ,
 Where $V_{t+1} \geq 0.27 \text{ m}^3$, BW (kg) = 984 V_{t+1} + 24.

Early estimates of energy needs

During the 1880s, Müntz & Grandeau in Paris and Wolff and colleagues in Hohenheim and, during the 1930s, Ehrenberg and his colleagues in Breslau, all measured the energy requirements of horses for maintenance and work by feeding defined rations and recording changes in body weight. In 1911 these data were supplemented by extensive respiratory experiments in Berlin by Zuntz and in Bonn by Hagemann and subsequently in 1931 by Fingerling in Mökkern (Klingeberg-Kraus 2001) and by Brody (1945) in Missouri, who recorded results in his classic text *Bioenergetics and Growth*.

Two general methods of estimating heat evolved in maintenance

In the absence of work achieved or other useful output, e.g. milk or weight gain, all the metabolic activity of maintenance results in heat evolved. This is measured through direct calorimetry by the absorption of heat in a water jacket and by collecting expired moisture. Alternatively, indirect calorimetry requires measurements of respiratory exchange and those of urinary N excretion, when the reactions are neither endothermic, nor partially anaerobic, and the energy equivalent of O₂ is known.

Indirect calorimetry

Indirect calorimetry was first used by Lavoisier and Laplace. It is based on the fact that O₂ consumption and CO₂ production are normally closely correlated to heat production, as illustrated by the oxidation equation for one gram-molecule, mole, of carbohydrate:



180 g 134.4l 134.4l (s.t.p.)*

1 l = 1 dm³ or 10⁻³m³

Thus the consumption of 6 mols (6 × 22.4 = 134.4l) O₂ in the oxidation of 1 mol hexose yields 2837 kJ (or 11 O₂ yields from carbohydrate oxidation 21.1 kJ). Similarly, with the oxidation of mixed fat, 19.6 kJ are generated per litre O₂ and from the oxidation of mixed protein 20.2 kJ per litre O₂ consumed. As the heat equivalent of O₂ consumed varies with the nature of the substrate oxidised it is necessary to know the composition of the fuel mix (carbohydrate, fat, protein) oxidised. The amount of protein oxidised is calculated from urinary N excreted, assuming that protein contains 16% N and that all urinary N is derived from protein. Thus, protein approximately = N × 6.25.

The relative amounts of fat and carbohydrate oxidised are determined from the non-protein respiratory quotient (RQ), i.e. mols of CO₂ produced to mols of O₂ consumed. For carbohydrate this is:

$$6\text{CO}_2/6\text{O}_2 = 1.00$$

The RQ for mixed fat is 0.71 (for short-chain fats the RQ is nearer 0.8 and for long-chain fats it is nearer 0.7) and for mixed protein it is approximately 0.81.

The RQ does not have the rigorous significance indicated above, as huge amounts of CO₂ are produced in the GI tract by anaerobic bacterial fermentation and by liberation of CO₂ from bicarbonates. (It will be noted that the heat of bacterial fermentation, as in ruminants, can help compensate for heat lost in cold climates and thus the horse may avoid a shivering response in maintenance.) Excess CO₂ may also be liberated under conditions of acidosis and over-ventilation and CO₂ may be stored during alkalosis. So the rate of O₂ consumption is the better measure of heat production. Furthermore, the energy equivalent of CO₂ has a relatively wide range, whereas the range for O₂ is relatively narrow over the possible range of RQ. Finally, the RQ of protein corresponds to the average energy value of O₂, 20.2 kJ/l. From these arguments the conclusion is drawn that under normal conditions the practical method of measuring energy metabolism requires knowledge only of the rate of O₂ consumption, corrected to standard conditions. Heat production is calculated by assuming an equivalent energy value for O₂ of 20.2 kJ/l. This corresponds to the RQ of 0.82 in accord with the evidence in Table 6.1 (below).

* Standard temperature and pressure are the conditions the volume of dry gas would assume at 0°C and 760 mm pressure.

Table 6.1 Constants at s.t.p. for protein, fat and carbohydrate when oxidized in the animal body (Brouwer 1965).

	Carbon %	Consumed on oxidation of 1 g		Set free on oxidation of 1 g			Respiratory quotient (RQ)
		O ₂ g	O ₂ l	CO ₂ g	CO ₂ l	Heat kJ	
Protein*	52.00	1.366	0.957	1.520	0.774	18.4	0.809
Fats**	76.70	2.875	2.013	2.810	1.431	39.7	0.711
Starch	44.45	1.184	0.829	1.629	0.829	17.6	1.00
Saccharose	42.11	1.122	0.786	1.543	0.786	16.6	1.00
Glucose	40.00	1.066	0.746	1.466	0.746	15.6	1.00

*The approximate composition of protein is: N: 16%; C: 52%; 23.8kJ/g.

** It is assumed that fats contain only a small proportion of short-chain fatty acids.

Supplementary information:

- molar volumes O₂, N₂, CO₂, CH₄, H₂ = 22.41 l;
- heats of combustion: CH₄ = 39.54 kJ/l;
- H₂ = 12.76 kJ/l

From Table 6.1 The values below follow:

- Protein (g) = 6.25 × N g;
- Carbon (C) in protein (g) = 3.25 N g; and
- Fat (g) = 1.304 × C g

Using constants given in Table 6.1, energy balance (E) may be calculated from C (g) balance and N (g) balance:

$$E \text{ (kJ)} = 51.831C - 19.40N$$

Heat production (H) determined from measurement of gas (O₂, CO₂ and CH₄) exchange

Heat production (H kJ) from the net volume of oxygen (O l) consumed at s.t.p., the volume of carbon dioxide (C l) produced at s.t.p., methane production (M l) at s.t.p. and the mass of urine nitrogen (N g) may be expressed as:

$$H \text{ (kJ)} = 16.18O + 5.02C - 2.17M - 5.99N \quad (1)$$

(NB Include carbon dioxide occurring in the urine as free CO₂ and as carbonate.)

The CO₂ and O₂ are determined in the flow and pressure of the incoming and outgoing air. The range in concentration is CO₂: 0.390–1.003% and O₂: 19.981–20.589%. The carbon content of feed, faeces and urine is determined. The contribution of urine to the C balance is only about 1/10 of that from faeces, but the coefficient of variation % for urine is ten times larger than that for faeces (Thorbeck *et al.* 1965). [For the determination of the heat of combustion of urine it is necessary

to use urine acidified with H_2SO_4 , not in unnecessarily large excess, in order to avoid loss of carbon and this carbon must be taken into account (Nijkamp 1965). HCl should not be used]. Moreover, in respiration experiments, the chemically bound CO_2 in urine must be determined for the calculation of the total production of CO_2 .

Heat production determined from measurement of O_2 exchange only

In experiments in which O_2 consumption only is determined, heat production can be estimated with sufficient accuracy on the assumption that the heat equivalent of oxygen is 20 kJ/l. This heat equivalent implies that the RQ is 0.75 on the Brouwer (1965) scale. The value for heat equivalent when the RQ is 0.81 is 20.24, but rounding to 20 is considered admissible.

$$H \text{ (kJ)} = 20 O \text{ (l)} \quad (2)$$

CONCENTRATES AND ROUGHAGES

All horses should receive some form of long roughage, such as fresh grass, hay or silage. A proportion of this may be replaced by succulent green or root vegetables and soaked sugar-beet pulp. Hay or grass may comprise the total ration of idle horses and usually forms at least half the ration. The concentrate portion of the daily feed may therefore vary from nothing to 50%, and only in exceptional circumstances, or in the hands of experienced feeders, should it rise to the proportions of three-quarters of the daily allowance of dry feed.

Concentrated feeds, such as cereals, cereal by-products, oilseed meals and the like, are traditionally fed by the bowl, that is, by volume. The energy and nutrients that these feeds provide are, of course, much more closely associated with their weight than with their volume and feeding containers should thus be calibrated to show the volume occupied by a unit weight of each type of feed. Table 5.5 (p. 131) contains average conversion values for cereals, although it will be appreciated that the bushel weight of cereals varies from season to season and from crop to crop, according to how well they were grown. The energy content per kilogram of each type of concentrate also differs. Ideally, therefore, feed bowls should be calibrated to indicate the volume, giving multiples of 2 MJ DE (or alternatively 1 MJ NE), for each type in use.

FEED ENERGY

The gross energy of a feed is the heat evolved when it is subjected to complete combustion in an atmosphere of oxygen. Obviously, all this energy, measured as heat, is not available to the animal because a portion of the feed remains undigested and is voided in the faeces. In addition, a relatively unknown quantity is lost from the horse as the gases methane and hydrogen, in the main by passage out through

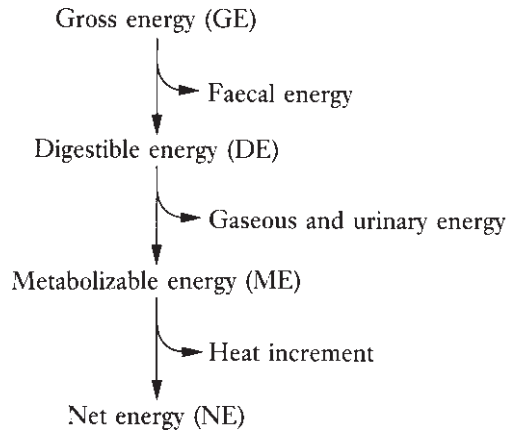


Fig. 6.5 Partition of dietary energy.

the anus, but also by absorption into the blood and exhalation. Of the products of digestion and fermentation that are absorbed, a proportion of the amino acids is deaminated and the nitrogen incorporated in urea. Much of this is excreted in the urine.

The gross energy of a feed, less the energy content of the faeces attributable to it, is the digestible energy (DE). Subtracting the energy content of combustible waste gases voided, and urine excreted, leaves the metabolizable energy (ME). This is the residue of feed energy that is available to the body for its various processes of tissue repair, the functioning of organs, the physical work of skeletal muscles, growth and milk production. The efficiency of ME utilization depends on the precise chemical form of the nutrients derived from the diet and on which of these functions is performed. The efficiency is measured either as the amount of useful product or from the quantity of waste heat dissipated. The ME less this heat increment attributable to the feed is the net energy (NE). (Heat increment is the heat loss of a nourished animal in excess of that lost by a fasting animal.) The scheme is summarized in Fig. 6.5. NE is used for maintenance, growth, work, reproduction, etc.

When energy demands are great, concentrate feeds, such as cereal grains, must form part of the diet if those demands are to be met, simply because the horse can consume larger quantities of dry matter daily when cereals are included and they contain more ME per kilogram of dry matter. Conversely, an idle horse has relatively low energy requirements, yet its appetite should be satisfied. As the horse can consume daily lesser quantities by weight of bulky fibrous feeds than of concentrates, then its appetite is more likely to be satisfied with lower intakes of energy when fibrous feeds are used. This idle horse is a stabled, normally active, nonworking animal, described as having energy requirements for maintenance only, that is, those leading to a zero change in body weight, or, more accurately, a zero change in body energy content.

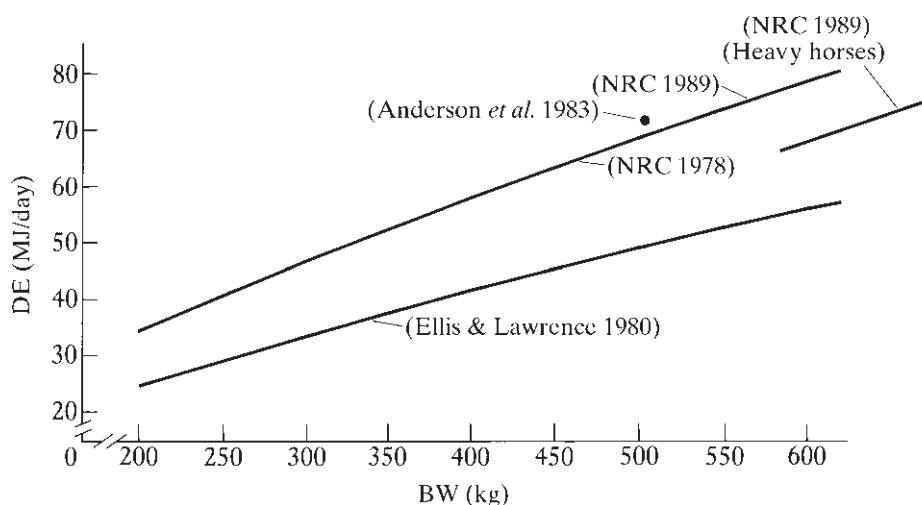


Fig. 6.6 Approximate relationship of kg BW to daily DE requirements for maintenance according to four sources: for weights 600 kg BW or less, $DE \text{ (MJ/day)} = 5.9 + 0.13 \text{ BW}$ (NRC 1989), for weight >600 kg BW, $DE \text{ (MJ/day)} = 7.61 + 0.1602 \text{ BW} - 0.000063 (\text{BW})^2$ (NRC 1989); $DE \text{ (kJ/day)} = 649 \text{ BW}^{0.75} \equiv 0.649 \text{ BW}^{0.75} \text{ MJ/day}$ (NRC 1978); $DE \text{ (kJ/day)} = 465 \text{ BW}^{0.75} \equiv 0.465 \text{ BW}^{0.75} \text{ MJ/day}$ (Ellis & Lawrence 1980).

NRC (1989) assessment of maintenance needs of adult horses

The energy requirements for maintenance per 100 kg body weight decline slightly with increasing body weight, so that relative to body size larger horses require slightly less food for maintenance than do ponies in similar conditions. This may account for the first term in the equations below, and, to compensate for it, ponies may develop a greater barrel or appear more pot-bellied. The relationship between body weight (BW) and the DE requirements for maintenance is depicted in Fig. 6.6. Two of these curves are derived from the identities formulated by the NRC (1989):

$$DE \text{ (MJ / day)} = 5.9 + 0.13 \text{ BW} \quad (3)$$

where BW is the body weight (kg) of a normal nonworking horse weighing 600 kg or less. Work at the Texas Agricultural Experiment Station indicated that cutting horses, working in a hot environment, expend 10–20% more energy than would be predicted (Webb *et al.* 1990).

Texas workers (Potter *et al.* 1987) found that the energy requirement of heavy (675–839 kg) Belgian and Percheron horses was 10–20% lower than predicted by Pagan & Hintz (1986a). The difference was attributed to the lower activity of heavy horses and the slower rates of acceleration and deceleration during voluntary work. Hence, the NRC (1989) made an adjustment for heavy horses:

$$DE \text{ (MJ / day)} = 7.61 + 0.1602 \text{ BW} - 0.000063 \text{ BW}^2 \quad (4)$$

where BW is the body weight (kg) of horses in excess of 600 kg, as these engage in somewhat less voluntary activity (partly accounting for the negative quadratic term in the equation).

Maintenance needs of growing horses

The daily maintenance requirements of growing horses, determined by extrapolation of growth data to zero gain, were found to be 158 kJ DE/kg BW and 148 kJ DE/kg BW for limit- and for *ad libitum*-fed horses, respectively (Cymbaluk *et al.* 1989a). (In the same study 24–83 g BW gain/MJ DE was achieved above maintenance.) This is about one-quarter of the efficiency of poultry and pigs. The values of 148–158 kJ agree with equation (3) for animals of 200–300 kg BW, but clearly can be applied only to animals within the BW range studied by Cymbaluk and colleagues. Individual horses differ in their needs about all these means. Some will eventually become fat when subjected to a regime under which others will lose condition.

Heat production and efficiency of ME use

An inactive horse at the maintenance level of energy intake and expenditure does essentially no work on its surroundings, so that NE expended in maintenance (m) is ultimately degraded to heat:

$$ME_m = NE + HI = \text{heat production at maintenance} \quad (5)$$

where HI is heat increment, or waste heat. The fact that the temperature of the horse's body is normally greater than that of the surroundings, to which heat is continuously being lost, is the expression of this situation. Exposure to a cold or a wet and windy climate accelerates metabolic rate so that the rate of heat production keeps pace with the rate of heat loss in order to maintain a steady body temperature, that is, the energy requirements for maintenance rise.

Conversely, in hot climates, where the environmental temperature is higher than that of the horse, the heat produced must still be dissipated. This is done primarily by evaporation of sweat and of water from the lungs, but also by a rise in body temperature. A physiological stress is induced. Thus, in one environment heat production is a boon and in the other a hindrance. Work at the Texas Agricultural Experiment Station (reviewed by Hiney & Potter 1996) indicated that cutting horses, working in a hot environment, expend 10–20% more energy than would be predicted by the equations of Pagan & Hintz (1986b). Their rectal temperatures were often 41°C, and one may assume that, as metabolic rate is a function of body temperature, this was a cause of the greater need.

Can heat production be manipulated to the horse's advantage? Waste heat (HI) is a measure of the efficiency of utilization of the ME of feed and it is known to vary between types of feed. If the NE available represents 80% of the ME ($NE/ME = 0.8$), then the remaining 20% is HI. When feeds are selected for use, their difference

Table 6.2 Estimated efficiency of utilization by the horse of ME (NE/ME), or k , for various energy sources.

	For maintenance* (k_m)	For fat deposition (k_f)
Mixed proteins	0.70	0.60
Meadow hay	0.70	0.32
Lucerne hay	0.82	0.58
Oats	0.83	0.68
Barley	0.85	0.77
Fat	0.97	0.85

*These values are higher than those for fattening mainly because the use of these nutrients for that purpose spares the breakdown of body fat.

in HI should ideally be considered in the context of the climate and the purpose for which the horse is kept. Allowance for these differences is the basis of the justification for the French (INRA) NE system, discussed later in this chapter (see The NE system introduced in France by INRA 1984, updated 1990).

Some estimates of the likely efficiency of ME utilization by the horse are given in Table 6.1 and Fig. 6.7. The efficiency values (k values) in Table 6.2 subtracted from 1 show the proportion of energy lost as waste heat when the feed is used for maintenance or for fat deposition. Thus, 30% of the energy of meadow hay would be lost as waste heat by horses at maintenance, whereas only 15% of the ME of barley would be similarly lost (note that the utilized energy is ultimately degraded to heat also, but more hay would be required for maintenance). During winter, ample meadow hay may be a more appropriate feed than in the summer, or than barley, as the greater HI of hay may contribute to the maintenance of body temperature when the weather is cold.

The partition of the GE of four feeds is described in the histograms in Fig. 6.7. It should be clear that k represents, in the main, efficiency of glycogen and of depot fat formation. Efficiency of utilization of these sources by muscles is approximately 0.35–0.45. The concept of work measured for flat racing etc. is illusory, as the true efficiency can be measured only as a difference in energy expended between exercise on the level and that on a gradient, not on an inclined moving belt. When a horse moves up a gradient, work is done against the force of gravity, whereas on an inclined moving belt the horse does not rise. It remains at the same level. However, it is known that, for other reasons, a horse expends more energy running on an inclined belt than when on a horizontal belt moving at the same speed. Robert *et al.* (2000) showed that the electromyographic activity of hind-limb muscles during a trot increased with increasing slope of the treadmill at inclinations of 0%, 3% and 6%, implying an increasing workload.

The k value includes an allowance for the energy costs of both ingestion and digestion, that is the energy expended in eating, digesting and fermenting feed, in addition to differences amongst nutrients in the systemic efficiency of their metabolism in ATP, tissue, milk etc. synthesis (see Appendix C for maintenance k_m

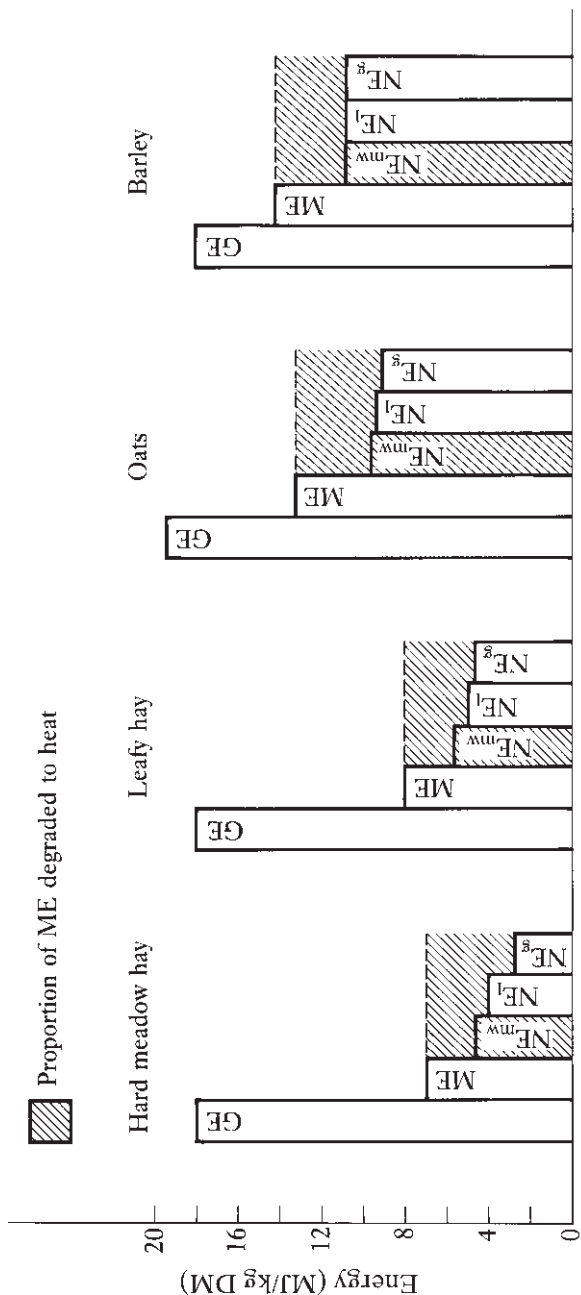


Fig. 6.7 Estimated average utilization efficiency of gross energy (GE) of feeds for several production purposes: NE_{mw} – NE for maintenance, or for work, which may in practice be approximately the same; NE_l – NE for lactation; NE_g – NE for growth.

Efficiency of utilization of GE as a source of ME = $ME/GE = q$
Efficiency of utilization of ME as a source of NE = $NE/ME = k$
 $NE/GE = qk$

calculation). Note: nearly two-and-a-half times as much hard hay is required for maintenance as would be required of barley so that nearly 25% more heat is produced at maintenance on the hay diet.

The ribs of both breeding and working horses in optimum condition cannot be seen, but can be felt with little fat between the skin and ribs. Acclimatization to cold weather does not necessitate excessive fat deposition but should allow sufficient time for the coat to grow. Horses should therefore be provided with a shelter protecting them from rain, snow and the worst of the wind. In other words, three sides and a roof provide sufficient protection in all seasons for properly fed adult animals. (The author has successfully held horses in this way at temperatures lower than -30°C .) A long hair coat, if dry, and a modicum of subcutaneous fat are an excellent insulation for horses given an ample roughage diet, so that the rate of waste heat production without shivering, a function of $(1 - k)$, equates with the rate of heat loss. In the spring when horses are brought in, daily grooming and 57–114 g oil added to the ration each day should accelerate the shedding of the winter coat.

Joyce & Blaxter (1965) determined the heat loss to the environment of sheep, measuring the insulation properties of the skin and fleece. The external insulation (I_E) of sheep is the resistance to heat flow from the skin surface to the environment and that resistance depends particularly on the temperature difference between the skin and the air and the length of the fleece. External insulation of sheep was shown to be linearly related to fleece length (Joyce & Blaxter 1965). The situation in the horse is somewhat similar. Booth *et al.* (1998) used indirect calorimetry to determine whether soaking the full winter coat of two mature Shetland pony stallions of 178 and 200 kg BW with water of 5.26°C caused a change in the rate of heat production. They were housed at an ambient temperature of 2.0 – 9.5°C in an open sided shed and given meadow hay at maintenance levels. Skin temperature decreased, but not rectal temperature, and heat production was not increased over three hours (also see Chapter 10). In the absence of shelter, ambient air movement may increase and so this will increase the rate of heat loss.

Production needs

The measurements of Anderson *et al.* (1983) given in Table 6.3 indicate the energy needs for maintenance plus endurance work. Figure 6.8 gives the DE requirements of horses of various weights at maintenance and when engaged in work of a range of intensities, strenuous work causing a large increase in energy demand. Excessive HI, or waste heat, in working horses is an encumbrance and a contributory cause of unnecessary sweating, indicating an important attribute of concentrate feeds, in addition to that of meeting the high energy demands of working animals. These demands of high productivity, also exemplified by peak lactation, are met from two main sources: (1) the breakdown of body fat and (2) increased feed energy.

The data in Table 6.2 show that in growing/fattening animals 68% of the ME of meadow hay and 40% of the ME of mixed proteins are lost as waste heat, whereas

Table 6.3 DE demands for maintenance plus work on a slope of 90° at endurance rates (135 heart beats/min, 155 m/min).¹

Body weight (kg) ²	400	500	600
Distance travelled (km)	DE per day (MJ)		
1	68	79	90
2	76	88	100
4	89	103	117
6	99	114	127
8	107	121	133
Approximate appetite	108–113	125–130	140–145

¹Based upon a quadratic equation relating energy requirements to body weight and work in Quarter Horses (Anderson *et al.* 1981, 1983).

²Average body weight 503 kg.

only 23% of barley grain and 15% of fat ME are similarly lost in fattening. The so-called heating effect of cereals and other concentrates reflects a more rapid rise in blood glucose and metabolic rate after a large meal and the associated feeling of vigour in 'hot-blooded' breeds (see also Calming effects of dietary fats, Chapters 5 and 9). In Fig. 6.9 the interactions among the HI of feeds, environmental temperature, and production of body heat and critical temperature are depicted.

Partition of feed energy

Visual examination of feed reveals nothing about its ME content, but the feed can be weighed. Fortunately, the gross energy (GE) content of most horse feeds is just over 18 MJ/kg DM. This statement is untrue for feeds containing much more than 80 g ash/kg or 35 g oil/kg. For example, oats on average may contain 45 g oil and 19.4 MJ GE/kg DM. Figure 6.7 shows how the GE of samples of four different feeds might be utilized for growth or for hard extended work and the data give a revealing and objective comparison of roughages with cereals. The coefficient q represents the approximate efficiency by which the GE of each feed is utilized as a source of ME and as the coefficient k represents the efficiency by which this ME is utilized for the functions of maintenance, growth etc., $q \times k = \text{NE}/\text{GE}$, or the overall efficiency of utilization of the 18 MJ for the productive function.

Note that $q \times k$ of the hard meadow hay for growth (mainly fat deposition) is 0.12, whereas the equivalent for barley is 0.59, a value 4.9×0.12 . The NE in hard meadow hay for growth, and probably for extended work, is only a quarter of that found in the two cereals, despite a similarity of their GEs. In energy terms, that is, MJ/kg feed, the losses in the utilization of ME from hay and cereals are not very different (Fig. 6.7), but the k values are (Fig. 6.10), for the reason that the ME values (MJ/kg) differ widely.

It is recognized that roughages are required by horses and ponies, particularly in a long form, in order to maintain general metabolic health and a feeling of

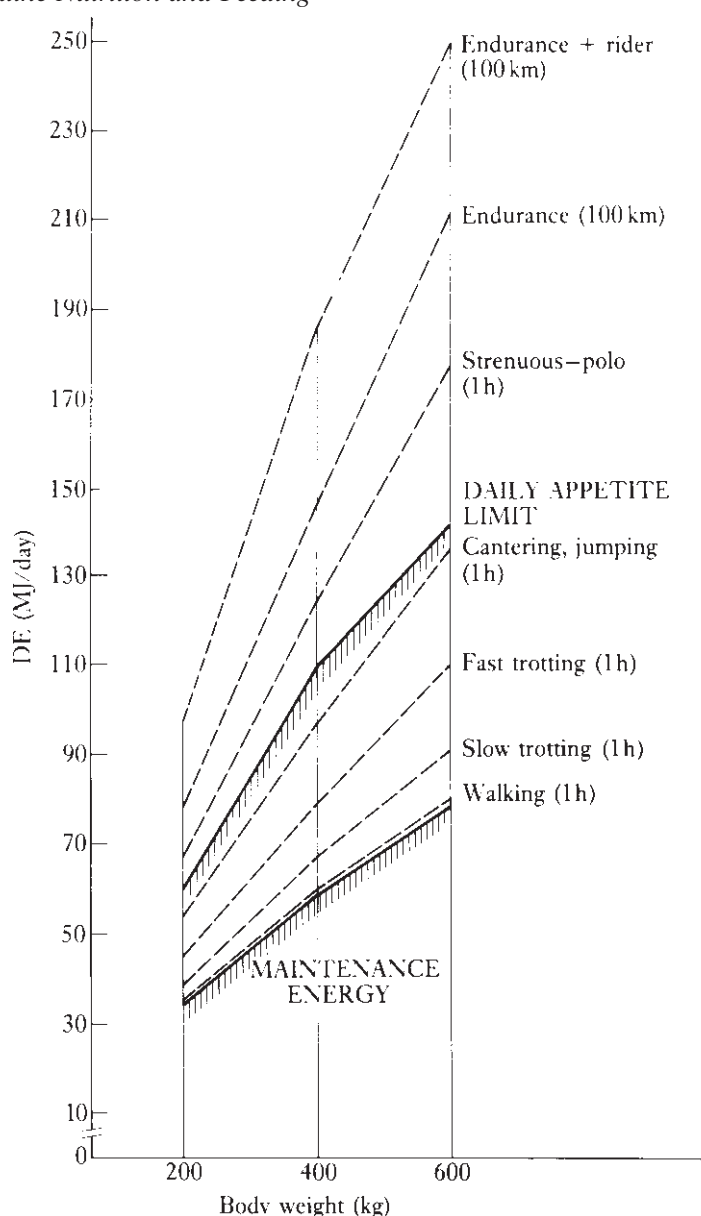


Fig. 6.8 DE demands of daily maintenance and work at a constant elevation in relation to appetite of horses of three body weights. (Effect of a 67 kg rider on 400 and 600 kg horses and 33 kg rider on 200 kg horse given for endurance rides only.)

well-being. However, are there lessons to be learnt from the above calculations? First, poor-quality roughages can be an expensive buy if they are to form a major part of the ration of growing or hard-worked animals. Second, hard-worked animals can lose condition if poor-quality roughages form a major portion of their diet. Finally, idle horses may put on unwanted fat if too much cereal is included in their diet.

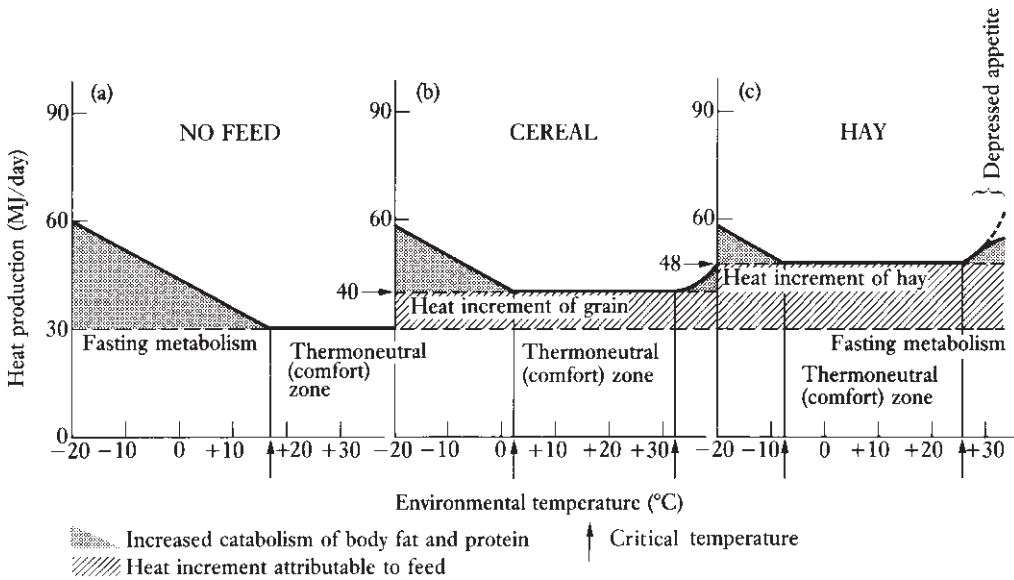


Fig. 6.9 Interactions among the heat increment of feeds, environmental temperature, heat production and critical temperature. Data are approximate; they assume minimal air movement and are not based on direct experimentation. (a) Fasted horse (also with slightly less subcutaneous fat); (b) horse fed 3.5–4 kg grain providing 40 MJ ME and 30 MJ NE (maintenance level); (c) horse fed 5–6 kg hay providing 48 MJ of ME and 30 MJ of NE (maintenance level).

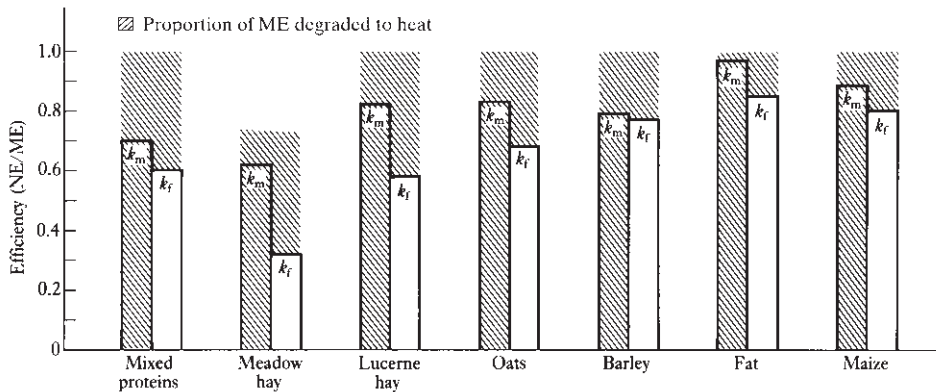


Fig. 6.10 Estimated efficiency of utilization of ME (NE/ME), or k , for various energy sources: k_m = NE/ME for maintenance; k_f = NE/ME for fat deposition (later growth). (Maintenance values are higher than those for fattening, mainly because the breakdown of body fat is spared by the use of these feeds for maintenance.)

The data available on the ME content of horse feeds and on their NE for various functions is limited (but estimated values for maintenance are provided by INRA and selected data are given in Appendix C expressed as horse feed units, UFC). The large difference in the efficiency of DE utilization for productive activity between roughages and concentrated feeds led Martin-Rosset and colleagues (1994) to develop the UFC NE system (see ‘The NE system’ introduced in France by INRA

1984, updated 1990, this chapter). For proper use of the DE system it is necessary to follow certain rules (see 'Ration formulation using the DE and NE systems', this chapter) and to use DE data provided in Appendix C.

DIGESTIBLE ENERGY (DE), PROTEIN AND MINERAL REQUIREMENTS BASED ON NRC RECOMMENDATIONS

Reproduction and lactation

The dietary requirements of the breeding mare can be arbitrarily divided into those for: (1) the first eight months of gestation; (2) the last three months of gestation; and (3) lactation (this may coincide with 0–4 months of gestation). Gestation length for most TBs is in the range 335–345 days and for other breeds 322–345 days. Lactations of 110–130 days are typical of many husbandry systems, although the nonpregnant mare would produce milk for much longer if given the opportunity.

Energy

The first eight months of gestation have no practical impact on nutrient needs – that is, they do not raise requirements above those for the barren mare, nor do they increase the already high requirements of the lactating mare. Thus, after weaning, this mare's energy requirements approximate those of maintenance until eight months of gestation have been completed. Most of the foetal growth occurs during the last 90 days of gestation. Even so, the nutrient drain incurred then to sustain normal foetal and placental growth is much less than that for lactation. The approximate energy contents of the foetus and the other products of conception at term, compared with the energy content of the mare's milk over a four-month lactation, are given in Table 6.4. By assuming that all foetal growth occurs during the last 90 days, the approximate DE required daily above maintenance to meet these needs can then be calculated (Table 6.5) for comparison with the much greater demands of milk production.

As the foetus occupies an increasing proportion of the mare's abdominal cavity, her capacity for bulky feed declines during the period in which nutrient requirements increase. This may correspond to an increase in the quality of grazing (see Chapter 10), but, where mares are given hay and concentrates, the quality of the

Table 6.4 Approximate energy contents of the foetus and other products of conception at term compared with the energy content of mare's milk over a four-month lactation.

Mare's weight (kg)	200	400	500	600
Products of conception at term (MJ)	110	200	240	270
Lactation of 17 weeks (MJ)	1700	2840	3400	3900

Table 6.5 DE required daily to meet the needs of foetal growth and lactation, but excluding maintenance requirements for energy of the mare.

Mare's weight* (kg)	400	500
Products of conception at term (MJ)	5.0	6.0
Average milk production (MJ)	40	50

*The mare's weight should increase by 15% during gestation so that her maintenance requirements rise proportionately.

forage should be improved during the last three months of gestation. The energy and nutrient requirements of the breeding mare (Table 6.6) increase from period (1) to (2) and from (2) to (3) of the cycle given above. (Note that, if the mare is lactating during part of period (1), her requirements then will exceed those of period (2).) However, the values are averages and the amounts of feed and therefore the DE given to individual pregnant mares should be adjusted to avoid either obesity or poor condition (see also Chapter 7).

During the 11th month, the DE requirement is equivalent to a 0.33:0.66 mixture of oats and hay of nearly 10kg daily. Part of the demand in the 11th month is to sustain udder development. On average, these values differ little from the flat-rate recommendations of Table 6.6. Feeding studies have not established whether birth weight of the foal is generally influenced by deviations from those rates.

One investigation (Goater *et al.* 1981) demonstrated an increase of both 1.5kg in birth weight and 0.24kg in daily weight gain during the first 30 days of life as a result of providing the mare with 120% of the NRC (1978) gestation rates. Other experiments, in which Quarter Horse and TB mares were restricted to 55% and Arabian mares to 85% of the rates, led to weight losses by the pregnant mares without affecting birth weight of the foal in comparison with mares receiving the recommended rates. Clearly, healthy mares possess the capacity to adapt without the foal incurring any significant handicap.

Protein and minerals

The most critical nutrients for breeding mares given traditional feeds are protein, Ca and P. Mares kept during the last 90 days of gestation entirely on reasonably good-quality pasture or high-quality conserved forage containing some 30–40% leafy clover, lucerne or sainfoin require no other source of Ca, and, if the forage contains 10% protein per unit of dry matter, no supplementary protein.

An increase in the physiological demand for Ca leads to a decrease in serum total and ionized Ca concentrations and a stimulation to parathormone secretion. This occurs in periparturient mares during mammary Ca secretion when serum total Ca has been shown to decrease from 3.1 to 2.7mmol/l (12.5–11mg/dl) (Martin *et al.* 1996a). The evidence indicated that the dietary requirement of the mares averaging 510kg BW in late pregnancy was closer to 5.5g Ca/kg dietary DM (45g Ca/day) than

Table 6.6 Daily DE requirements of horses for various functions and amounts of hay and concentrates needed to provide the energy (based on NRC 1989 recommendations).

Mature body weight	200 kg			400 kg			500 kg			600 kg		
	DE (MJ)	Hay ¹ (kg)	Concentrate mixture ² (kg)	DE (MJ)	Hay ¹ (kg)	Concentrate mixture ² (kg)	DE (MJ)	Hay ¹ (kg)	Concentrate mixture ² (kg)	DE (MJ)	Hay ¹ (kg)	Concentrate mixture ² (kg)
Mature horse ³ maintenance	31.0	3.8	—	56.1	6.8	—	68.6	8.4	—	81.2	9.9	—
Mares last 90 days of gestation	35.6	2.9	1.1	64.9	5.1	2.0	79.5	6.1	2.6	94.5	7.6	2.8
Lactating mare first 3 months	64.0 (12) ⁴	2.6	3.8	101.2 (18) ⁴	4.7	5.5	122.2 (21) ⁴	6.0	6.4	141.0 (23) ⁴	7.2	7.2
Lactating mare 3 months to weaning	51.0	3.4	2.0	82.4	5.9	3.0	102.0	7.5	3.6	120.9	8.7	4.3
Stallion breeding	38.9	2.2	1.9	70.3	3.4	3.7	85.8	4.3	4.4	101.7	5.2	5.2
nonbreeding	35.0	2.8	1.1	62.0	4.7	2.0	75.0	5.8	2.4	89.0	6.9	2.9
Weanling (6 months old)	35.0	1.0	2.4	57.3	1.8	3.7	67.4	2.2	4.3	75.7	2.3	5.0
Yearling (12 months old)	39.7	2.2	1.9	68.2	3.6	3.4	84.1	4.4	4.2	100.0	5.1	5.1
Long yearling (18 months old)	37.5	3.1	1.1	69.0	5.0	2.5	87.9	6.2	3.2	104.6	7.5	3.8
Two year old excluding work	33.0	2.7	1.0	64.0	4.6	2.3	78.7	5.8	2.8	98.3	6.9	3.7
maintenance plus 1 h moderate work	47.7	2.5	2.4	90.0	4.6	4.6	110.1	4.8	6.2	135.1	5.3	8.0

¹ Hay containing 8.2 MJ DE/kg and 88% DM.
² Concentrate mixture containing 11.4 MJ DE/kg and 88% DM. Quantities of concentrate up to 1.5% BW daily may be fed if a minimum roughage allowance of 1 kg/100 kg BW is given.
³ 2.3 kg extra feed daily should produce 0.5–0.6 kg gain.
⁴ Assumed peak daily milk yield (kg).

to 3.5 g Ca/kg DM when the Ca was derived from bluegrass–clover pasture and concentrates, a third of the daily Ca being derived from the pasture. The P requirements, however, amount to 3 g/kg (0.3%) of the total dry diet. This would be provided by good pasture (see Table 10.2, p. 369), but as both grass and legume hays given to horses in the UK normally contain less than 2 g P/kg (0.2%), a supplement of dicalcium phosphate, or wheat bran will be required. The discrepancy would be met daily by a supplement of 60 g dicalcium phosphate or 1.5 kg bran for horses and 40 g or 1 kg, respectively, for ponies. Where grass hays are used, as assumed in Table 6.6, supplementary protein will be required as well. If the hay contains 7% of protein and it constitutes 70% of the diet, whereas the diet as a whole should contain 10% protein, then the concentrate must contain 16–17% of protein (Appendix A shows the type of calculation needed). This is the level found in most commercially prepared stud nuts that would also provide the necessary P, vitamins and trace elements.

Abundant good-quality pasture will meet the energy, protein, Ca and P needs of lactation, even though the minimum dietary protein requirement will have risen to 125 g/kg dry feed (12.5%). Responses in milk yield have been obtained from Quarter Horse mares by providing mixed feeds containing up to 170 g protein/kg. However, grass and clover proteins are of high quality and it is unlikely that economic responses would be obtained by raising the protein level of the spring-grazing diet. If the stocking density is high, or good-quality pasture is otherwise moderately limited, supplementation can be provided by lower protein pony nuts (see Table 7.1, p. 249), or a mixture of these and cereals. If pasture is more scarce, a stud nut or an equivalent mix containing 16–17% of protein should be given to lactating mares. Any conserved forage provided should be leafy hay containing a mixture of clover and grass, or should be well-conserved haylage. Typically in the UK, grass hays of only moderate quality are fed when grazing is limited. These contain only 40–80 g crude protein/kg and so do not meet immediate needs.

It is unlikely that one could overfeed lactating mares with roughage, except that large quantities of poor roughage would limit their capacity for concentrates, leading to a decrease in milk yield. When typical grass hays are given, a satisfactory milk yield is obtained only if at least 50% of the dry feed is provided as a stud nut or equivalent 16–17% protein mix. This mix may be based on oats or barley and soya-bean meal, or an equivalent proprietary protein concentrate containing 440 g protein/kg. Horse mares would require 1.75 kg of soya daily (12–14% of the total grass hay and cereal-based ration). The reason why a 16–17% protein mix is sufficient for lactating horses, as well as for late pregnancy, despite a higher protein requirement in lactation, is that the mix forms over half the ration in lactation to meet the greater energy needs, whereas it forms only 30% of the prefoaling diet. The amino acid composition of milk is given in Chapter 7, under 'Lactation', from which Wickens *et al.* (2002) calculated amino acid requirements, assuming a digestibility of 65%. For a typical lactation these requirements are approximately three times those of maintenance. Milk protein is richer in leucine, isoleucine, threonine and valine compared with skeletal muscle protein; and from muscle composition the authors

calculate the maintenance requirement for each amino acid. However, these calculations assume a similar availability and muscle turn-over efficiencies for each amino acid, which are probably unjustified assumptions.

This grass hay, cereal, soya diet must be supplemented with a mineral mix composed of 35 g dicalcium phosphate, 65 g limestone and 70 g sodium chloride when the total daily intake of dry foods is 14 kg. Proportionately less will be required for smaller rations. A proprietary mixture of trace elements and vitamins should also be given. The latter should include vitamin A for horses with no access to pasture. Where large amounts of silage or haylage are used, supplementary vitamins D and E will also be necessary at levels, respectively, of 7000 iu and 250 mg daily. Whereas the trace-element content of mare's milk (but see 'Trace elements', Chapter 3), and therefore the adequacy of the foal's diet, is affected by the supplementation of the dam's diet, a deficiency of water, energy, protein, Ca or P will ultimately bring about a decrease in milk output, without altering its composition.

Growth of the foal

As horses grow they do not simply increase in weight and size, they also display what is termed development. Various tissues and organs of the body grow at different rates. In proportion to body size the rate of weight gain of the body as a whole, if permitted by the feed allowance, is very much greater in the younger than in the older animal. In fact, from the suckling period onwards the rate of gain per 100 kg BW declines continuously, but the rate of growth of long bone and muscle declines at an even faster rate. An increasing proportion of the gain constitutes fat, which has much higher demands for feed energy. These trends are fundamental to a formulation of requirements for protein, Ca and P in particular, which decline fairly rapidly as a proportion of total diet with increasing age of the foal and yearling (see Tables 3.3, p. 60, and 6.7). Further details of growth, and the way in which we should guide it, and indeed, of the breeding mare, are given in Chapters 7 and 8.

Table 6.7 Nutrient concentration in diets for horses and ponies expressed on the basis of 90% DM (based on NRC 1989).

	Crude protein (g/kg)	Ca (g/kg)	P (g/kg)
Mature horses and ponies at maintenance	72	3.2	2.0
Mare, last 90 days of gestation	94	5.5	3.0
Lactating mare, first 3 months	120	5.5	3.0
Lactating mare, 3 months to weaning	100	4.0	2.5
Creep feed	160	8.0	5.5
Foal (3 months old)	160	8.0	5.5
Weanling (6 months old)	135	6.0	4.5
Yearling (12 months old)	115	5.0	3.5
Long yearling (18 months old)	105	4.0	3.0
Two year old, light training	95	4.0	3.0
Mature working horse, light to intense work	95	3.2	2.0

RATION FORMULATION USING THE DE AND NE SYSTEMS

Both the DE and NE systems require the accumulation of two sets of information:

- (1) nutrient content of feeds (Appendix C); and
- (2) nutrient requirements of horses described in the *same* terms as for feed (Table 6.6 for DE).

These two issues are covered in the text and tables.

Energy needs

The principle that has been adopted in calculating the daily requirements for many nutrients is to assume that the needs of various functions of the horse are additive, i.e. a factorial system has been used, the factors or functions being maintenance, work, growth, reproduction etc. This approach is not precisely supported by biological evidence, but it is the simplest approach and the values (coefficients) can be modified and the factors augmented as new information comes to hand, or as the activities of horses are extended. The daily energy needs for:

- maintenance (m) are a function of BW;
- work (w), a function of BW, intensity (I) and time (T) spent;
- growth (g), a function of weight gain (G) and BW relative to mature size;
- pregnancy (p), a function of the mare's BW and stage of pregnancy (S_p); and
- lactation (l), a function of stage of lactation (S_l) and yield per day (Y).

Rationing (kg feed per/day) is based upon energy allowances, and in the case of a pregnant, lactating mare, energy requirements would be summated from:

$$m(BW) + p(BW \times S_p) + l(S_l \times Y) = \text{energy needs (MJ / day)} \quad (6)$$

which, when divided by the energy content of the feed (for that energy system), gives the kg feed per day. Alternatively, appetite (defined as kg feed per day) would need to be estimated and then the required energy density of the diet calculated from:

$$\text{needs(MJ)} / \text{appetite(kg)} = \text{MJ / kg diet}$$

The proportions of roughage and concentrates are then calculated:

$$\text{MJ / kg diet} = x(\text{MJ / kg roughage}) + (1 - x)(\text{MJ / kg concentrates}) \quad (7)$$

where x is the dietary fraction or proportion (typically 0.6–0.7) of roughage and the remainder of the diet ($1 - x$) is concentrate (therefore typically 0.4–0.3), ignoring water. This gives the proportions of roughage and concentrate making up the total appetite (kg/day).

The formulation of a ration requires estimates of: (1) total daily dry food intake, (2) the energy content of feeds, and (3) daily energy requirements (DE system, Tables 6.6 and 6.8). The two energy systems proposed for use, supplying information on (2) and (3), are the NRC DE system and the INRA NE system. The justification for each is that for:

- DE – digestibility is the most potent factor that segregates feeds that otherwise have similar GE values;
- NE – roughages and concentrates can be clearly segregated according to the efficiencies (k) by which the ME is utilized for maintenance and productive purposes.

(Note: the $q \times k$ values will differ somewhat according to the function of the horse, maintenance, fattening, milk secretion etc., however, for simplicity in application, the INRA system assumes that efficiencies (k) of ME for maintenance apply to all functions. Moreover, the energy requirements for growth account for only 20% of the total energy requirements of the growing animal (Vermorel & Martin-Rosset 1997). The remaining 80% is consumed in maintenance. In the final analysis, the practical value of each system depends importantly on the reliability of its feed-evaluation system (Appendix C). Precise nutritional definitions are elusive for roughages and succulents. Nevertheless, the relative value of the two systems in practice will depend on the development of this evaluation.

The DE system

The DE required in MJ/day divided by dry feed intake in kg/day gives MJ of DE per kg of dry feed needed. The DE contents of roughages and concentrates are given in Appendix C and their required dietary proportions can then be roughly calculated using equation (7) above, examples of which are given in Appendix A. Equation (7) has been used in deriving the proportions in Table 6.9. The greater the intensity of physical activity, the higher the proportion of cereals required. As the speed of the horse increases, the energy expended rises steeply on an hourly basis (Table 6.8). Hence, the types of problem encountered can be quite different in horses undertaking strenuous effort compared with those asked to respond in a leisurely fashion. Furthermore, compared with ponies, large horses tend to require a higher proportion of concentrates in the ration when both are subjected to hard work.

The recommendations in Tables 6.6 and 6.9 are likely to be somewhat in error where low energy densities are required by working horses. In those situations the feed energy requirements are likely to be underestimated and so the French NE system is discussed below.

The NE system

The NE system was introduced in France by INRA in 1984 and updated in 1990.

Table 6.8 DE demands of maintenance and work on the flat (based on NRC 1989).

Body weight (kg)	200	400	600
Approx. feed capacity per day (MJ DE)	60	100	150
Maintenance requirement per day (MJ DE)	31	56	81
	Energy requirements for work above maintenance (MJ DE)*		
Walking (1 h)	0.4	0.8	1.3
Slow trotting, some cantering (1 h)	4.2	8.4	12.5
Fast trotting, cantering, some jumping (1 h)	10.5	20.9	31.4
Cantering, galloping, jumping (1 h)	25.0	50.0	75.0
Strenuous effort, racing, polo (1 h)	36.0	72.0	108.0
Slow trotting, some cantering (10.4 h, 100 km) calculated from above	43.5	87.0	130.5

*1 kg concentrate provides about 12 MJ DE.

Table 6.9 Effect of a range of required energy densities (MJ DE/kg air-dry feed) on the cereal content of the daily ration when hays of two energy contents are available.

Energy density of ration required	Oats (%)		Barley (%)	
	7.2 ¹	7.8 ^{1,2}	7.2 ¹	7.8 ^{1,2}
7.5	7	0	5	0
8.0	19	5	14	4
8.5	30	19	23	14
9.0	42	32	32	24
9.5	54	46	41	34
10.0	65	60	50	44
10.5	77	73	59	54
11.0	88	86	68	64

¹Energy content of hay (MJ DE/kg): 7.2 MJ/kg, medium quality; 7.8 MJ/kg, good quality.

²Hay can be assumed to contain 86% DM and where haylage of 45% DM is to be used it may be substituted for the hay of 7.8 MJ DE in the proportions 1.8–1.9 kg haylage per 1 kg hay. Similarly, 1.6–1.7 kg haylage of 50% DM could be used.

Energy

The NE system first provides the NE content of feedstuffs for maintenance. The feed values in this scheme are expressed in unitless horse feed units (UFCs), i.e. relative to a reference value of 1 for barley, where 1 kg standard barley has an NE value of 9.414 MJ (assuming barley contains 140 g moisture/kg). Thus:

$$1 \text{ UFC} = 9.414 \text{ MJ NE} \quad (8)$$

In the INRA tables, and Appendix C, UFCs are given per kg DM, so that UFC values are: barley 1.16, maize 1.33 etc. (i.e. each determined UFC value is divided by its fractional DM content). For barley, with 86% DM, the value is $1/0.86 = 1.16$.

Energy value of feed

The NE contents of feeds for *maintenance* are calculated from their ME contents and the coefficients of their respective efficiencies of utilization for maintenance:

$$\text{NE} = \text{GE} \times \text{dE} \times \text{ME/DE} \times k_m \quad (9)$$

where dE is the digestibility calculated from the digestibility of organic matter (OM) and k_m is NE/ME for maintenance.

The UFC of a feed is its NE value relative to that of standard barley:

$$\text{UFC} = (\text{ME} \cdot k_m) / 9.414 \text{ (as per equation 8)}$$

where NE of barley is 9.414 MJ/kg and ME is in units of MJ/kg (NB UFC is unitless).

Maintenance was chosen as it represents 50–90% of energy expenditure of horses and the NE feed values for maintenance are considered by INRA to be equivalent to those for physical activity, or work, a common equine function (i.e. $k_m \equiv k_w$). The NE for both maintenance and activity is expended mainly in ATP synthesis. In using the system one should apply both the INRA feed *and* requirement values.

UFC values were chosen as a basis of feed formulation for two main reasons: first, they are approximately additive, that is, different combinations of feedstuffs yielding the same total UFC should have the same productive effect, or the UFC value of a feed is not influenced by other feeds with which it may be combined. Martin-Rosset & Dulphy (1987) showed that, in the horse, digestibility of feed was also not influenced by feeding level, and that the digestibility of forage was not affected by the addition of concentrates to the ration, in contrast with the effects in sheep. (The variable that has the largest influence in the discrimination of feedstuffs is dE, which is accounted for in both DE and NE feeding systems.)

The second, and most important, reason for selecting NE is that k_m draws a clear distinction between the productive values of forages and concentrates. The energy costs of mastication and propulsion of digesta through the GI tract and the heat of fermentation of forages in the hind-gut are greater than the heats of ingestion and digestion of starch. In addition, the efficiency of utilization of VFA derived from forage fermentation is less than the efficiency of glucose metabolism from starch. These costs and efficiencies both affect the value of k_m . In other words, the formulation of mixed feeds from the DE values of their constituent ingredients, with the objective of deriving a variety of mixtures with the same productive energy values, exaggerates the value of forages.

The k_m value of standard barley is 0.79 and that of an average grass hay is 0.62. The following comparisons (Table 6.10) between concentrates and forages on a DM basis will exemplify the point. The ratios of the means indicate that, relative to concentrate values, the DE system overvalues these roughages by 15% for produc-

Table 6.10 Comparison of the DE and NE values for concentrates (C) and forages (F) per unit DM.

	DE (MJ/kg)	UFC
Barley (C)	15.2	1.163
Maize (corn) (C)	16.1	1.35
Oats (C)	13.4	1.01
Maize silage (F)	11.2	0.88
Grass hay (F)	7.3	0.53
Barley straw (F)	6.8	0.28
Ratio F/C	0.566	0.480

Table 6.11 Energy cost of eating as a proportion of the ME value of the individual feed measured at 1.26 times maintenance (Vernet *et al.* 1995).

	Proportion of ME/kg feed DM expended in eating
Pelleted maize	0.010
Pelleted sugar-beet pulp	0.042
Long hay	0.102
Wheat straw	0.285

tive purposes. However, the large effects occur only with materials at the extremes, i.e. maize and barley straw, otherwise the bias introduced by the DE system (ingestion only) is small (see Table 6.11). INRA have provided data to support the adoption of the UFC system, and the conclusions they draw are in line with proposals put forward by Frape and Tuck (1977).

Energy cost of eating

Chewing, ingestion and digestion of feed involve muscular activity. In addition, there is an increase, above the fasting rate, in general metabolic rate. Vernet *et al.* (1995), using indirect calorimetry in respiration chambers, measured the energy expended by sport horses during ingestion of feeds of different types. These feeds were measured at the level of 1.26 times the maintenance energy requirement, when maintenance energy demands were increased by an average of 38.8% during eating a mixed meal, and heat production (maintenance) is increased by 75–95% during a meal of straw or hay (Vermorel & Martin-Rosset 1997). For single feeds the energy costs of eating per kg DM intake are given in Table 6.11.

The energy cost of eating is mainly accounted for by the energy expended in chewing. An additional amount is contributed by cephalic activation of the sympathetic and parasympathetic nervous systems. In other words, the sight and smell of feed stimulates a physiological response of the whole GI tract and of some other organs. The measurements by Vernet *et al.* included only the increments in expenditure during actual eating and so excluded much of, for example, the HI of bacterial

fermentation. The data do indicate that with poor-quality feeds, such as straw, over a quarter of the ME they contribute is lost during the process of taking the feed into the stomach. For mixed feeds, the energy costs of eating are 6–10% of ME (Vermorel *et al.* 1997), and although the horse expends a similar amount of energy to that used by ruminants per minute it expends two or three times as much as the ruminant per kg DM, owing to a lower rate of eating (Vermorel & Martin-Rosset 1997).

Protein

The scheme also accounts for the digestible crude protein (DCP) content of feeds, and in the case of forages these are reduced in proportion to their NPN contents. The useful protein is calculated from the estimated amounts of amino acids absorbed from the small intestine (plus amino acids absorbed from the large intestine) and this useful protein is termed MADC (*matières azotées digestibles corrigées*) (g/kg DM) (see forage K values [not to be confused with the coefficient *k*] in 'Protein and the evaluation of N absorbed from the large intestine', this chapter – see below).

ENERGY AND PROTEIN REQUIREMENTS BASED ON INRA FEED UNITS, EXPRESSED AS UFC AND MADC

The recommended allowances in UFC for particular physiological functions were determined by a factorial method, or by feeding experiment. The scheme therefore partly allows for differing *k* values for maintenance, growth, lactation and work (NB INRA assume the same *k* value applies for maintenance and work – see 'Energy value of feed', above) for each feedstuff. Thus, *k* values partly depend on the particular mix of nutrients, that is, the substrate for metabolism, i.e. VFAs, fats, glucose and the proportions, or balance, of the amino acids (see Appendix C). In these respects, the variation in *k* is greater for growth than for maintenance. Products of poor-quality roughage have relatively less value for growth than for maintenance. Although *k* values differ for different functions, UFC values will differ less as they are unitless ratios.

Maintenance

The maintenance requirement of an idle horse is:

$$0.038 \text{ UFC / kg BW}^{0.75}$$

or 4 UFC/day for a gelding weighing 500 kg.

The maintenance requirement of working horses, cf. horses at the maintenance level of activity, cf. 'easy list', is assumed to be increased by 5–15% and that of stallions

is increased by 10–20%. The higher maintenance heat production assumed for working horses results from a higher metabolic rate, and may account for the difference in actual feeding rates of racehorses compared with their needs calculated in DE (see Chapter 9).

Protein for maintenance of adult horses

Dietary protein required for maintenance of adult horses may be of poorer quality than that needed for growth and so, within the range of dietary proteins given to horses, no adjustment for amino acid balance is needed in the requirement proposed by INRA. Daily protein requirement for maintenance of an adult horse is:

$$2.8 \text{ g MADC / kg BW}^{0.75} \quad (10)$$

so that a 500 kg horse requires 295 g MADC daily.

Protein for maintenance related to DM intake and endogenous faecal N losses

The horse needs protein, which is digested, yielding amino acids (and probably dipeptides) which are absorbed. The apparent digestibility of protein differs only to a small extent between sources. Apparent digestibility is expressed as:

$$\frac{(\text{N intake} - \text{faecal N})}{\text{N intake}}$$

Meyer (1983b) concluded that precaecal apparent N digestibility was 0.5–0.6. As this is ‘apparent’ it makes no allowance for endogenous N secreted into the lumen of the GI tract, whereas true digestibility does. Mean true values should be slightly greater than this range indicates and true protein digestibility in the small intestine ranges from 0.45 to 0.80. Endogenous faecal N loss is proportional to body weight:

$$\text{Daily endogenous N} = 52 \text{ mg} \times \text{kg BW}^{0.75} \text{ (Slade } et al. 1970)$$

although endogenous loss also varies with the level of feeding. This may account for the much higher estimate of Meyer (1983b) of 180 mg N/kg BW^{0.75}, giving:

$$\text{Endogenous N} = 3 \text{ g N / kg DM intake} \quad (11)$$

At constant DM intake, apparent N digestibility increases as the N content of the diet is increased, because the endogenous loss forms a smaller proportion of the total N. Likewise, true digestibility, for which the endogenous N loss is subtracted from the faecal N, is similar for a range of dietary N concentrations. For most protein sources, true digestibility falls in the range 0.7–0.9, and no allowance should be made for any change in dietary N concentration.

Protein sources that contain anti-nutritive factors can increase endogenous protein secretions. In the pig, toasted field-bean protein, which may still contain

some anti-nutritional factors, leads to a negative apparent N absorption in the small intestine (ileal N digestibility), although true protein digestibility is reasonable. If this were to apply to the horse, it would have two consequences:

- (1) the measurement of endogenous N production using protein-free diets may seriously underestimate endogenous secretions; and
- (2) if only nonamino-acid N is absorbed from the large intestine, then amino acids contained in endogenous protein secretions passing the ileocaecal valve would be lost to the horse and the stimulation to secretion by anti-nutritional factors would compromise feeding value.

A consequence of this is that field-bean protein may have a much lower amino acid value than field-pea protein, despite similar apparent N digestibilities and not too dissimilar true protein digestibilities. A conclusion concerning the horse may not yet be drawn. It should be noted that whereas apparent N digestibility reflects the net absorption of N to be metabolized by the horse, true digestibility does not, and requirements in that case must also be in terms of truly digested amino acids. On the other hand, apparently digested N will over-estimate usable N because a major proportion of that N absorbed from the hind-gut will be NPN from which the horse gains no material benefit.

Endogenous urinary N

Horses given an N-free diet will continue to lose N in the urine, through the metabolism of tissue proteins. This is taken to be the minimal endogenous renal N loss, and was provisionally calculated (Meyer 1983b) as:

$$\text{Daily endogenous renal N} = 165 \text{ mg N} \times \text{kg BW}^{0.75} \quad (12)$$

There is renal conservation of N in that endogenous urinary loss is minimal in horses on low-N diets and the amount increases with an increase in dietary protein, whereas endogenous faecal N loss is relatively constant with a change in dietary protein content.

Endogenous integumental N

Losses of N from skin and hair should be allowed for. An average value of:

$$\text{Daily integumental N} = 35 \text{ mg N} \times \text{kg BW}^{0.75} \quad (13)$$

is taken (Meyer 1983b). This slightly underestimates the rate of loss during seasonal hair shedding.

Total endogenous N

A reasonable total endogenous N value from the three sources of loss is:

$$\text{Daily basal endogenous N} = 380 \text{ mg N} \times \text{kg BW}^{0.75} \quad (14)$$

(i.e. $180 + 165 + 35$), equivalent to 2.4 g crude protein.

N balance has been achieved during feeding trials with about 350 mg digestible N/kg BW^{0.75}. Evidence indicates that horses thrive better with some accumulation of protein reserve. Therefore, in crude protein (CP) terms the maintenance requirement (m) for DCP is taken to be:

$$\text{DCP}_{(m)} \text{ (g / day)} = 3.3 \text{ g} \times \text{kg BW}^{0.75} \quad (15)$$

(i.e. $\text{CP} = \text{N} \times 6.25$).

The energy requirement for maintenance according to German workers (Meyer 1983b), is:

$$\text{DE}_{(m)} = 0.6 \text{ MJ} \times \text{kg BW}^{0.75}$$

(in accordance with the INRA value of 0.038 UFC or $0.36 \text{ MJ NE} \times \text{kg BW}^{0.75}$).

So, in relation to energy, requirement for maintenance is:

$$5.5 \text{ g DCP/MJ DE}$$

or

$$9.2 \text{ g DCP/MJ NE} \quad (16)$$

Breeding mare

Pregnancy

The amounts of energy deposited in the products of conception (foetus + placenta + foetal membranes + udder) are approximately:

- 8th month, 0.636 MJ/100 kg BW/day; and
- 11th month, 1.954 MJ/100 kg BW/day.

In the 8th–9th, 10th and 11th months of pregnancy, respectively, 14%, 41% and 45% of the total energy at birth is deposited in these products (Table 6.12 and Table 6.6). The INRA system assumes an efficiency of ME utilization (k) for pregnancy of 0.25. However, the daily allowance in UFC is adjusted according to the mare's condition, as undernutrition of a mare in good condition has no adverse influence on the foal's

Table 6.12 Weight gain and composition of the foetus (Martin-Rosset *et al.* 1994).

Month	Weight gain (g/kg birth weight)	Foetus weight gain, fraction of birth weight	GE content (MJ/kg)*	Protein content (g/kg)
8	190	0.18	4.18	115
9	190	0.20	4.60	130
10	300	0.30	4.94	153
11	310	0.31	5.36	171

*Of conceptus (foetus + foetal membranes + uterus + udder).

Table 6.13 Mare's milk yield and composition (Martin-Rosset *et al.* 1994).

Month	Milk yield (kg/100kg BW)	Milk composition	
		Energy (MJ/kg)	Protein (g/kg)
1	3.0	2.41	24
2	2.5	2.09	24
3	2.5	2.09	24
4	2.0	1.99	21

birth weight, or on its growth rate. The effect of this adjustment on fertility at foal heat etc. has not been established.

Lactation

The mean characteristics of milk adopted in the INRA system are given in Table 6.13 (see also Table 7.2, p. 254). The efficiency of ME use (k) assumed in estimating feed requirements is 0.65.

Protein requirements

Prior to the eighth month of pregnancy, the mare's protein requirement does not materially exceed that for maintenance. Foetal growth, as a fraction of birth weight, increases each month from the eighth, when it is about 0.18–0.23 of that at birth (Table 6.12). In breeds weighing 500kg, mature weight:

$$\text{Foetal weight (kg)} = 0.00067x^2 - 20 \quad (17)$$

where x is time in days from fertilization (Meyer 1983b).

$$\text{Birth weight (kg)} = 0.45 \text{ BW}_m^{0.75} \quad (18)$$

where BW_m is mare's weight (kg) (Gotte 1972 in Meyer 1983b).

Equations 17 and 18 indicate that foetal growth rate accelerates towards the end of pregnancy and that birth weight is a function of mare's weight, i.e. small mares drop small foals. The mean birth weight of a foal out of a 500kg mare is 47.6kg.

The protein content of the foetus also increases from 115g/kg in the eighth month to 171g/kg at birth (Table 6.12) and the protein retention in foetal membranes, uterus and mammary gland is about 0.2 of that in the foal. The fraction of the total protein (a) retained in the foal at birth was estimated (Meyer 1983b), giving the additional daily protein requirement of a pregnant mare for foal's growth *in utero*:

$$\text{DCP (g/day)} = 6.15a(\text{BW}_m)^{0.75} \quad (19)$$

where BW_m is mare's weight (kg) (Table 6.14).

Table 6.14 Protein requirements of breeding mares (g DCP/day) (based on Meyer 1983b). These estimates differ from INRA tables (Anon 1984).

Month	Fraction (a) of total protein retained in foal at birth	Body weight of mare (kg)		
		200	500	800
<i>Pregnancy</i>				
8th	0.14	227	451	641
9th	0.22	250	500	700
10th	0.23	260	528*	740
11th	0.31	282	561	798
Last days	0.10			
<i>Lactation</i>				
1st		585	1163	1655
3rd		611	1214	1727
5th		468	931	1324

* For example, this value is the sum of 150 g for foetus (equation 19) + 30 g for membranes, etc. (0.2 of foetus) + 348 g for mare (equation 15).

Thus, the mare's dietary protein requirement increases from the eighth month of pregnancy as the fraction of the total protein (a) increases.

The milk production of mares increases up to about the third month, whereas its protein content decreases from about 24 g/kg milk in the first month to 21 g/kg in the fourth month (Table 6.13). Meyer (1983b) assumes that the efficiency of utilization of DCP for the products of conception and milk protein synthesis is 0.5, leading to the recommendations given in Table 6.14.

INRA protein requirements for pregnancy

For the growth of the uterus, including its contents, and of the udder, the amount of protein retained daily is 5 and 21 g/100 kg BW in the 8th and 11th months, respectively. INRA assumes a metabolic efficiency for DCP of 0.50–0.55. Thus, the extra concentrate protein required by a 500 kg mare is approximately 45–50 g at 8 months, and 190–210 g at 11 months (Table 6.15 values less mare's maintenance requirements; the values in this table have been updated in accord with more 1990 INRA data). These values are considerably lower than Meyer's values (1983b).

INRA protein requirements for lactation

The mean composition of milk is given in Table 6.13. Assuming a metabolic efficiency for DCP of 0.55, an INRA requirement of 44, 38, and 36 g DCP/kg milk in months 1, 2–3 and 4 of lactation, respectively, is derived [also lower than Meyer's estimates (1983b)]. This gives a requirement, including maintenance, of 950 g for a 500 kg mare in the first month, in agreement with the values quoted by Martin-Rosset *et al.* (1994), and approximately in accord with the revised values given in Table 6.15.

Table 6.15 Daily energy (UFC) and protein (MADC, g) requirements of horses as proposed by INRA [Anon 1984; updated by INRA, Ed. Martin-Rosset (1990)]. All figures in brackets are assumed average daily gain (kg).

Adult weight (kg)	450		500		600	
	UFC	MADC	UFC	MADC	UFC	MADC
Maintenance	3.9	275	4.2	295	4.8	340
Light work	6.6	450	6.9	470	7.5	510
Medium work ¹	7.6	515	7.9	540	8.5	580
Intense work ²	6.9	470	7.2	490	7.8	530
Mare, gestation						
month 8	3.8	315 ⁵	4.1	340 ⁵	4.7	395 ⁵
months 9–10	4.3	425	4.7	460	5.4	535
month 11	4.4	445	4.8	485	5.5	565
Mare, lactation						
month 1	8.2	865	8.9	950	10.5	1125
month 2	7.0	700	7.6	770	8.9	910
month 3	7.0	700	7.6	770	8.9	910
Growth ³						
6 months	4.2 (0.70)	500	4.5 (0.75)	530	5.1 (0.85)	600
8–12 months	5.1 (0.70)	560	5.5 (0.75)	590	6.2 (0.85)	660
20–24 months	6.3 (0.40)	380	6.8 (0.45)	420	7.8 (0.55)	480
32–36 months	5.9 (0.15)	300	6.5 (0.20)	330	7.6 (0.30)	390
Stallion ⁴						
resting	5.8	400	6.1	420	6.3	440
breeding	6.6–8.0	480–620	7.0–8.4	490–630	7.0–8.5	520–650

¹2 hours daily.²1 hour daily.³Based on equations: daily UFC/kg BW^{0.75} = a + bG^{1.4}, and daily MADC (g) = a BW^{0.75} + bG, where a = coefficient of maintenance, b = coefficient of gain, and G = body weight gain (kg/day) (see Table 6.16).⁴Including 0.5 hours medium exercise daily.⁵These values agree with Martin-Rosset *et al.* (1994) resulting from a deposition of 5 g/100 g BW and 0.5–0.55 metabolic efficiency for protein in products of conception.

Growth

The efficiency of ME use for growth has not been established and will depend both on the energy substrates and on the proportions of fat and protein laid down (NB The index of the second term in equation 20 probably derives from the increase in fat deposition resulting from faster growth rates). Maintenance accounts for 60%, or more, of the energy expended, depending principally on the rate of growth allowed. The total requirements are assumed to fit the following relationship:

$$\text{UFC/kg BW}^{0.75}/\text{day} = a + bG^{1.4} \quad (20)$$

where a = coefficient of maintenance requirement, b = coefficient of gain, and G = average weight gain (kg/day) (Table 6.16).

Assuming 1 kg gain at 250 kg BW, requirement = 5.5 UFC
(maintenance 3.6 + gain 1.9)

Table 6.16 INRA coefficients used in the estimates of requirements for growth of light breeds (daily UFC/kg BW^{0.75} = a + bG^{1.4}, and daily MADC (g) = a BW^{0.75} + bG).

Age (months)	UFC		MADC	
	a	b	a	b
6–12	0.0602	0.0183	3.5	450
18–24	0.0594	0.0252	2.8	270
30–36	0.0594	0.0252	2.8	270

where a = coefficient of maintenance requirement; b = coefficient of gain; G = average weight gain (kg/day).

Assuming 1 kg gain at 350 kg BW, requirement = 7.0 UFC
(maintenance 4.7 + gain 2.3)

Assuming 0.5 kg gain at 300 kg BW, requirement = 5.0 UFC
(maintenance 4.2 + gain 0.8)

Economy may be achieved by allowing the slower growth rate in winter on mixed feeds, followed by accelerated compensatory growth on pasture in the spring (see Chapter 8).

The protein requirements for growth are similarly calculated.

INRA requirements for growth: protein for growing foals

The rate of body-weight gain, as a fraction of mature weight, declines progressively with increasing age. Concurrently, the protein content of that gain decreases and the fat content increases as age increases. The protein content of each kilogram gained decreases from 197 g at 3–6 months to about 170 g at two years of age in horses of 500 kg mature weight. Breeds with lower mature weights grow faster, relative to their size, and reach near mature weights at a younger age than do larger breeds. With these considerations for growth in mind, the recommendations for DCP proposed by Meyer (1983b) are given in Table 6.17 (see Table 6.15 for INRA recommendations).

INRA protein requirements for maintenance of growing horses

The metabolic efficiency of DCP for growth is set at 0.45 and, as the amino acid turnover rate in growing horses is greater than in adults, so the maintenance requirement for MADC is higher during growth:

$$\begin{aligned} &\text{MADC maintenance requirement of growing horses} \\ &= 3.5 \text{ g/day/kg BW}^{0.75} \\ &[2.8 \text{ g MADC/day/kg BW}^{0.75} (21), \text{ for adults}]. \end{aligned}$$

Table 6.17 Digestible CP recommendations (DCP, g/day)* for growing foals at moderate and fast growth rates (after Meyer 1983b).

Age (months)	Weight of mature animal (kg) and growth rates									
	200		500		500		800		800	
			Moderate		Fast		Moderate		Fast	
	Average BW (kg)	DCP (g)	Average BW (kg)	DCP (g)	Average BW (kg)	DCP (g)	Average BW (kg)	DCP (g)	Average BW (kg)	DCP (g)
3–6	85	290	170	503	175	544	232	635	292	870
7–12	130	260	258	474	288	546	360	695	444	736
13–18	160	225	333	461	365	493	500	731	548	621
19–24	175	230	388	454	425	488	612	688	612	622

* Assumes requirements for maintenance of 4.5 g DCP/kg BW^{0.75} at 3–6 months, decreasing to 4.1 g DCP/kg BW^{0.75} at 19–24 months of age.

INRA lysine requirements for growing horses

Lysine is the limiting amino acid for growth in most conventional diets. The lysine requirement for growing horses is set at 6 g/kg dietary DM at 6 months, and 4 g/kg dietary DM at the yearling stage.

Work

INRA protein requirements related to energy requirement

(See also Chapter 9, where requirements are considered independently of N balance.) For work, the INRA recommendations are that protein needs are less than proportional to energy needs, whereas the NRC (1989) assumes that protein needs are directly in proportion to energy needs for maintenance and all amounts of work. Nevertheless, for simplicity, INRA recommends that the work of an adult horse above maintenance requires 60–65 g MADC/UFC, i.e. protein needs proportional to those of energy (Table 6.15), but growing horses in work would require more protein.

Protein is not wasted when chemical energy in muscle is transformed into kinetic energy. Energy for work is derived mainly from glycogen and free fatty acids. However, in extended hard work, blood urea concentration rises as a consequence of protein catabolism. Therefore protein requirements (Table 6.18) are generally considered to rise above maintenance needs to achieve N balance in working horses, accommodating:

- muscle protein anabolism;
- N losses in sweat of 1.4 g N/l (although urinary losses decrease);
- increased feed intake that increases endogenous faecal N; and

Table 6.18 Digestible CP requirements per kg BW^{0.75} in working horses (Meyer 1983b).

	Light work	Moderate work	Heavy work
DM intake (g)	70	80	100
Faecal N losses (mg)	210	240	300
N losses in sweat (mg)	12	30	50–145
DCP requirement (g)	3.7	4.1	5.3
DE requirement (MJ)	0.68	0.83	1.0
DCP/MJ DE (g/MJ)	5.4	4.9	5.3

- some muscle protein catabolism in hard work (adenine nucleotide cycling also increases in exercise, increasing uric acid excretion, see Chapter 9).

Thus, from Table 6.18 the protein requirements for work are taken to be 5.5 g DCP/MJ DE, in equation (16) for maintenance.

PROTEIN, MINERAL AND MICRONUTRIENT FEED VALUES AS DETERMINED BY THE INRA SYSTEM

Protein and the evaluation of N absorbed from the large intestine

The protein value of feeds is expressed as g DCP/kg DM, that is, dietary CP multiplied by the apparent digestibility coefficient. A proportion of the CP is converted to bacterial protein and the horse benefits only from amino acids that are absorbed, but of both bacterial and dietary origins. The proportion of DCP N that is amino acid N is less in green forages, and especially in silages, cf. soya and other concentrate proteins. This observation is reflected in the K coefficient by which DCP of forages is multiplied to give MADC (corrected digestible N matter), that is:

K = 0.90 for green forages
 K = 0.85 for hays and dehydrated forages
 K = 0.80 for straws and chaffs
 K = 0.70 for good grass silages
 in comparison with
 K = 1.00 for soya, cereals, etc.

$$\text{DCP} \times \text{K} = \text{MADC (g/kg DM)} \quad (22)$$

The absorption of amino acids takes place almost entirely from the small intestine. The absorption from the large intestine of amino acids released from forage and bacterial proteins may be of significance for horses receiving poor-quality forage

diets, adequate in fermentable energy and supplemented with NPN. However, INRA proposals (Tisserand & Martin-Rosset 1996) assert that amino acids synthesized within the large intestine do not materially contribute to the amino acid supply of the horse. Thus amino acid absorption from the hind-gut is not likely to be a significant factor for most diets. These conclusions are consonant with evidence from other sources referred to in Chapters 1 and 2.

The resolution of the issue concerning the absorption of amino acids from the large intestine is of practical significance. Bacterial amino acid synthesis in the hind-gut is considerable, and 50–60% of faecal N in the horse is accounted for by microbial protein (Tisserand & Martin-Rosset 1996). Cuddeford and colleagues (1992) reported that the apparent crude protein digestibility coefficient for high temperature-dried lucerne was 0.74 in comparison with a value of 0.36 for field-cured timothy hay, cut at full bloom (lucerne had twice the N digestibility of timothy).

Gibbs *et al.* (1988) showed that the precaecal N digestibility of high-protein lucerne hay was three-fold that of coastal Bermuda grass (in contrast to the data with sun-cured lucerne of Klendshoj *et al.* (1979) in Table 6.19), and the lucerne led to greater N retention. However, the net uptakes of N from the large intestine did not differ and the overall N digestibility of the lucerne was only 29% greater than that for the Bermuda grass hay. According to Schmidt *et al.* (1982) an amount of N equivalent to 25–30% of dietary intake flows from the ileum into the caecum, irrespective of feeding level or type. The important point is that if most of the N that is absorbed from the large intestine is ultimately lost in the urine, then the true differences in protein value between the lucerne and timothy, found by Cuddeford and colleagues, was much greater than two-fold. Some evidence indicates that a maximum of 10–12% of the plasma pool of amino acids is of microbial origin and derived from the hind-gut. Thus, the range in coefficients for N retention is much wider than that of true digestibility. Stated differently, where the true digestibility coefficient is low, the potential retention coefficient is very much lower, whereas with high true digestibility coefficients there is a smaller decrement in the potential retention coefficient (NB the actual retention depends on the tissue needs).

Table 6.19 True N digestibility (N g/kg N intake) in the digestive tract of the pony of mixtures of two ingredients providing equal amounts of CP and of hays (Klendshoj *et al.* 1979) in Martin-Rosset *et al.* (1994).

	CP intake (g/kg)	Small intestine	Large intestine
Bermuda grass hay + crimped oats	122	690	130
Bermuda grass hay + micronized oats	122	580	320
Bermuda grass hay + soya-bean meal	122	730	130
Bermuda grass hay	117	360	370
Lucerne hay	150	220	570
Lucerne hay	181	380	460

Table 6.20 CP (g) true digestibility coefficients of absorbable intestinal amino acids and MADC (g) (Jarridge & Tisserand 1984).

	Intake (g)		Small intestine	Large intestine		Digestion, total tract (g)	
	CP	Amino acids	True digestibility ¹	Entry ¹ (g)	True digestibility ¹	DCP	MADC
Concentrates	180	171	0.85	26	0.90	148	147
Spring grass	180	162	0.70	49	0.80	128	117
Barley-maize	110	105	0.85	16	0.90	90	90
Grass hay	110	99	0.50	49	0.75	65	54 ²
Grass silage	110	82	0.50	41	0.75	65	44 ²

¹ Of amino acids.² See K values in 'Protein and the evaluation of N absorbed from the large intestine', this chapter, for grass hay $65 \times 0.85 = 54$, for silage $65 \times 0.70 = 44$, thus allowing for the NPN content of the large intestinal true digestibility of 0.75.

Note that, in Table 6.20, the true absorption coefficient of amino acids for concentrates and for barley-maize was the same, i.e. 0.85, ($147 \div 171$ vs. $90 \div 105$); however, the amino acid balance of the cereals is likely to have been poorer, so in that case the potential N retention for growth would be lower for the cereals.

Calcium

(See also Chapter 3.) The bioavailability, or true digestibility, of dietary Ca varies considerably. The principal factors controlling this are:

- amount of dietary Ca (0.7 at requirement intake to 0.46 at several times requirement);
- amount of dietary P (10 g P added/kg diet containing 4 g Ca/kg reduced true Ca digestibility from 0.68 to 0.43);
- vitamin D status (of less significance in the horse than in some other domestic species);
- dietary oxalate and phytates (Ca:oxalate <0.5 causes NSHP; phytates bind Ca. Implicated oxalate-rich grasses include: napier, guinea grass, buffel, pangola, green panic, paragrass, kikuyu, setaria and probably some species of millet grass. Lucerne contains oxalic acid, but has a high Ca availability.);
- age of animal.

The major part of dietary Ca given to growing horses, is directed to bone calcification. Work by Lawrence *et al.* (1994) indicated that although maximum bone mineralization was reached by six years of age, 76% of maximum was achieved by one year. As bone growth is most rapid in the first year of life, the demand for dietary Ca is very pronounced during that time. There is some speculation that horses

predisposed to exercise-associated myopathies (see Chapter 11) have a temporary failure in their ability to control intracellular Ca concentrations. However, there is no evidence that supplementary dietary Ca, above the dietary requirement, has a prophylactic influence on the risk of myopathies.

Phosphorus

The bioavailability of plant P varies with the proportion present as phytates. Phytate P is digested by phytase (EC 3.1.3.8) present in the gut principally, or entirely, as microbial phytase. The digestibility varies between 0.25 and 0.35. The addition of yeast cultures to the diet has been shown to increase phytate P use, presumably by stimulating microbial activity in the hind-gut. Excess dietary P interferes with Ca utilization. Savage (1991) and Savage *et al.* (1993b) found that diets containing four times the NRC (1989) estimated requirement for P in growing foals, but the requirement for Ca, caused lesions of dyschondroplasia (OCD), without signs of NSHP (see Chapter 8). Cortical bone porosity was increased in the growing foals by the high P diet and the extent of osteoid-covered surfaces of cancellous bone decreased with time, despite a depression in growth rate. On the other hand, a diet containing over three times the NRC (1989) estimated requirement for Ca had no adverse effect.

Sodium, potassium, magnesium, trace elements and vitamins

Table 6.21 gives the requirements for vitamins, trace elements, sodium, potassium and magnesium, supplements of which should be unnecessary if a commercially prepared mixed feed is used at recommended rates. Where compounded feeds are not given, a proprietary mixture of trace elements and vitamins should be used, because in high concentrations such nutrients are toxic and the normal horse owner is unlikely to have facilities for their proper handling and weighing. Sometimes compounded feeds intended to form the entire concentrate portion of the ration are used as supplements to oats, so diluting their effects in so far as protein, minerals, vitamins and trace elements are concerned. Thus, a pellet providing 4000 iu vitamin A/kg, mixed 50:50 with cereals and in turn fed 50:50 with hard hay will provide approximately 1000 iu vitamin A/kg total diet. Dilution of this kind is frequently a cause of incorrect Ca:P ratios in rations.

Electrolytes

A discussion of electrolytes – sodium, potassium and chloride – is given in Chapter 9, where problems of hard training are tackled. The potassium and magnesium needs of normal activity should be automatically met where good-quality roughage is available. The sodium needs (Table 6.21) can be met by providing NaCl (common salt), for simplicity ignoring the contribution made by natural ingredients. Sodium comprises 40% of NaCl so that the NaCl allowance should be two-and-a-half times

Table 6.21 Minerals and vitamins per kg diet adequate for horses (based on NRC 1978).

	Adequate levels	
	Maintenance of mature horses	Mare, last 90 days of gestation, and lactating and growing horses
Sodium (g)	3.5	3.5
Potassium (g)	4.0	5.0
Magnesium (g)	0.9	1.0
Sulphur (g)	1.5	1.5
Iron (mg)	40	50
Zinc (mg)	60	80
Manganese (mg)	40	40
Copper (mg)	15	30
Iodine (mg)	0.1	0.2
Cobalt (mg)	0.1	0.1
Selenium (mg)	0.2	0.2
Cholecalciferol (vitamin D) (μg) ¹	10 (400iu)	10 (400iu)
Retinol (vitamin A) (mg) ²	1.5 (5000iu)	2.0 (6666iu)
D- α -tocopherol (vitamin E) (mg) ³	30	30
Thiamin (mg)	3.0	3.0
Riboflavin (mg)	2.2	2.2
Pantothenic acid (mg)	12	12
Available biotin (mg)	0.2	0.2
Folic acid (mg)	1.0	1.0

¹ 1 iu is equal to the biopotency of 0.025 μg cholecalciferol (vitamin D₃) or ergocalciferol (vitamin D₂).

² 1 iu is equal to the biopotency of 0.3 μg retinol (vitamin A alcohol). Grass carotene has 0.025 of value of vitamin A on a weight basis.

³ 1 iu vitamin E is the biopotency of 1 mg DL- α -tocopheryl acetate. Where 50 g supplementary fat of average composition is added per kg feed, the requirement rises to 45–50 mg α -tocopherol/kg, equivalent to 79–88 mg DL- α -tocopheryl acetate. Also see pp. 96, 97 for working horses.

Table 6.22 Characteristics of a good water source.

	mg/l
Ammonia (albuminoid)	<1.0
Permanganate value (15 min)	<2.0
Nitrite, N	<1.5
Nitrate, N	<1.0
Calcium	50–170
Lead	<0.05
Cadmium	<0.05
pH 6.8–7.8	
Total dissolved solids	<1000

the sodium allowance, that is, 8.75 g salt/kg provides 3.5 g sodium. In order that any excessive salt intake is satisfactorily counteracted, clean drinking water, free from contamination with salts, should always be available (Table 6.22), excepting the restrictions to its use discussed in Chapter 4.

Trace elements

The dietary allowances for trace elements, as distinct from those for the major minerals and electrolytes, do not change appreciably per unit of feed with a change in the function of the animal from growth to work or to various phases of reproduction. The secretion of iodide in mare's milk may slightly increase her basal needs for iodine, but the margin given in Table 6.21 should satisfy all needs. The difference between this allowance and the toxic level (the margin of safety) existing for selenium and iodine (the elements for which the difference in absolute quantities is probably least) is adequate in the hands of responsible individuals. However, it is unwise and potentially dangerous for the average horse owner to handle pure forms of trace elements.

Vitamins

The dietary allowances for the fat-soluble vitamins A, D and E per unit of concentrate feed (Table 6.21) again are not varied much per unit with a change in the function of the individual, bearing in mind also the capacity of the horse to store them. A reappraisal of the situation would be necessary if there was any radical change in the basic raw materials traditionally used for feeding horses, for example from dry cereal grains to root vegetables, or to high-moisture conserved cereals, the elimination of conserved pasture, and so on. An example of this is given for vitamin E and breeding mares (Chapter 4). Appendix B gives examples of dietary errors encountered by the author in practice. Such errors cover the whole range of dietary attributes.

SIMPLE RATION FORMULATION

The principal chemical components of a stabled horse's diet are depicted in Fig. 6.11. The amounts of protein, calcium and phosphorus needed per kilogram of ration are given in Table 6.7 and the levels in the concentrate-hay mixture can be calculated by multiplying their protein contents (g/kg) etc. (see Appendix C) by their proportions, and summing. Thus, for hard hay and oats:

$$55\text{ g} \times 0.6 + 96\text{ g} \times 0.4 = 71.4\text{ g protein/kg mixture}$$

If the protein requirement is 100 g/kg, then the discrepancy of 28.6 g can be made good by substituting some soya-bean meal (other suitable protein sources may be substituted in inverse proportion to their lysine contents) for some of the oats, but attributing the soya (or other protein) with a protein content that is the difference between its content and that of oats, that is:

$$440\text{ g} - 96\text{ g} = 345\text{ g protein (see Appendix C)}$$

Then the proportion of soya to include and of oats to remove per kilogram is:

$$28.6/345.0 = 0.083, \text{ i.e. or } 8.3\% \text{ or } 83\text{ g/kg mix}$$

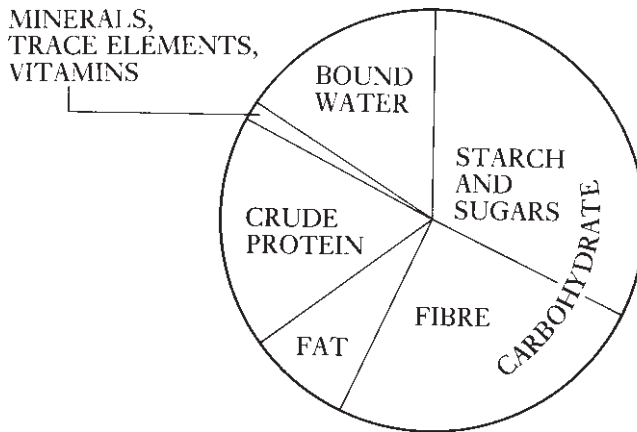


Fig. 6.11 Principal components by weight of a stabled horse's diet (excluding drinking water).

For simplicity, the effects of this substitution on the energy, calcium and phosphorus contents of the mix can be ignored. A similar calculation using Appendix C, and detailed in Appendix A, will have been adopted for calcium and phosphorus, but the deficits can be made good by adding limestone flour and/or dicalcium phosphate without making any substitution as for soya/oats. A calcium source of this type will normally be essential unless large amounts of lucerne or other legume forage are provided.

An approximate value for the DE content of a feed might be derived from its proximate composition, using the equation derived by Pagan (1997):

$$\text{DE MJ/kg DM} = (0.0886 + 0.510\text{CP} - 0.392\text{ADF} - 0.160 \text{ hemicellulose} + 1.974 \text{ fat} + 0.851\text{NSC} - 1.100 \text{ ash}) \text{ g/kg DM, } R^2 = 0.88 \quad (23)$$

NB: NDF = hemicellulose + ADF

Relationships derived by the author incorporate NSC and NDF values (see Chapter 2):

$$\text{DE kJ/kg DM} = (17.5 \text{ CP} + 36.5 \text{ EE} + 17.6 \text{ NSC} + 6.6 \text{ NDF}) \text{ g/kg,} \\ \text{for diets in which diet CF is 110–130 g/kg DM} \quad (24)$$

Comparable relationships were derived for other CF values.

Comparison of the DE and NE systems for ration formulation

Exact comparisons between the DE and NE systems are not possible from tabulated data because a definitive description is not provided for the feeds selected, so an assumed identity of feed composition between systems may not be assured. Moreover, the assumption made by the author that hay and barley had the same effect on capacity for feed is not justified, but was necessary for a simple comparison to be made in Table 6.23. Several general conclusions are reached:

Table 6.23 Application of DE (NRC) and NE (INRA) systems (kg/day) to the formulation of simple daily rations based on moderately poor grass hay, barley grain and extracted soya-bean meal. Mean requirement values of a horse, 500 kg at maturity, for energy and protein only, are used with feed intakes (kg DM/day; NRC 1978). Some of the outcome values are impractical. However, the purpose of the table is to compare outcomes for similar functions. See Table 6.24 for assumed analytical values of feeds used.

Growth (intake/day, kg)		DE system	NE system	Exercise (intake/day, kg)		DE system	NE system
Foal							
weaning (4.2)	Hay	0.33	—	Light (12)	Hay	11.60	9.36
	Barley	3.18	—		Barley	0.40	2.64
	Soya	0.69	—		Soya	—	—
Foal							
6 months (5)	Hay	0.93	1.05	Moderate (12)	Hay	9.48	8.03
	Barley	3.35	3.59		Barley	2.52	3.97
	Soya	0.72	0.36		Soya	—	—
12 months (6)	Hay	0.71	1.92	Protracted (12)	Hay	5.25	8.96
	Barley	4.61	3.68		Barley	6.39	3.04
	Soya	0.68	0.40		Soya	0.36	—
18 months (6.5)	Hay	0.07	0.99	Light (10)	Hay	7.90	6.27
	Barley	5.58	5.51		Barley	2.10	3.73
	Soya	0.85	—		Soya	—	—
24 months (6.6)	Hay	0.56	1.14	Moderate (10)	Hay	5.79	4.93
	Barley	5.40	5.46		Barley	4.21	5.07
	Soya	0.64	—		Soya	—	—
Mare							
maintenance (7.45)	Hay	5.30	5.92	Protracted (10)	Hay	1.55	5.87
	Barley	2.15	1.53		Barley	7.79	4.13
	Soya	—	—		Soya	0.66	—
gestation	Hay	4.04	5.02				
last 90 days (7.35)	Barley	3.01	2.16				
	Soya	0.31	0.17				
lactation	Hay	4.06	4.85				
0–3 months (10.1)	Barley	4.93	4.80				
	Soya	1.11	0.45				
3–6 months (9.35)	Hay	4.74	4.30				
	Barley	4.22	4.66				
	Soya	0.39	0.39				
Stallion (10)	Hay	7.91	5.20				
	Barley	2.09	4.80				
	Soya	—	—				

- the expected selection against poor hay by the NE system did not materialize;
- the NRC (1989) DE system generally seems to assume higher requirements of both energy and protein for the functions examined, and this counterbalances a relatively higher value of the usable energy in hay assigned by DE; and
- a change in the assumed feed intake of exercising horses had a large effect on the composition of the mixture selected by both systems.

The NE system should ultimately lead to economies in the selection of roughages for horses, as analytical work enlarges the fund of data on raw materials. These data

should predict the extent of precaecal digestibility and the energy yield during microbial digestion. The relative complexity of the NE system indicates that adjustments for a variety of husbandry practices may be accommodated. However, direct comparison of the two systems under several practical environments is required to determine their comparative effects on work, lactation, pregnancy, growth, health etc.

The ME of a feed is its DE less the associated energy lost in urine and that lost from the intestines as combustible gases (principally hydrogen and methane). On average, somewhat less than 20% of the energy apparently digested is lost to the horse by excretion in equine urine and by expulsion as methane. Vermorel *et al.* (1997) estimate that, with normal diets, horses lose about 4.2% of the GE of diets (8.0% of DE) in urine, but obviously greater amounts are lost with high protein diets metabolized as energy sources. Methane energy loss, owing to the activity of the cellulolytic and methanogenic bacterium *Archaea methanogenesis*, amounts to 1.95 SE 0.45% of GE in horses (Vermorel *et al.* 1997) and it may be assumed that a loss of energy amounting to about 30–65% of that lost as methane is lost as heat of fermentation (Webster *et al.* 1975). These losses differ amongst feeds, and losses during intermediary metabolism in tissues differ as between the nutrients, glucose, VFA etc. The ratio ME:DE is approximately 0.85 for grass hays and 0.90 for mixed diets in horses (Vermorel *et al.* 1997) and *km* % values vary from about 60–80 amongst feeds in the horse (Vermorel & Martin-Rosset 1997). Thus the efficiency of DE use for maintenance varies over a range of approximately 50–70% in the horse. The practical value of an NE system to replace a DE system depends substantially on this variation being reliably estimated amongst feeds that are available at economic prices.

Computers and diet formulation

(This section has been written with the assistance of John C. Dickens.)

During the past 30 years, computers, with an array of programmes of increasing power, have been used in the formulation of commercial feed mixtures. Programmes of lesser complexity are also available for use on the home personal computer. For this development to be truly successful it is requisite that several needs are met. These are that:

- (1) the nutrient requirements of the animal are reasonably well established;
- (2) the nutrient content and cost of a wide range of available feed materials are known and that the nutrient contents apply to the batch of each material available to the current user;
- (3) the needs for, and nutrient and non-nutrient characteristics of, roughages to be used are accommodated in any formulation. [By subtracting the nutrient content of the forage portion of the ration from the total requirement (per kilogram of total feed) the remainder gives the required nutrient content of the concentrate portion.] This procedure makes assumptions concerning the appe-

- tite of the horse (kg DM/day) discussed earlier (see 'Relationship of capacity for feed to body weight', 'Energy needs' and 'Appetite', this chapter);
- (4) physical, non-nutrient chemical and microbiological characteristics of all feeds concerned are known; and
 - (5) the effects of the characteristics in (3) and (4) on each class of horse are understood and allowed for during formulation.

Where this information is comprehensive it is possible to generate the formulae of mixtures that should be safe, nutritionally adequate, acceptable to the average horse and economical to use.

Principles of least-cost feed formulation

The number of chemical characteristics of each feed material that is accommodated in a formulation programme depends on its power. The simplest takes into account protein (g/kg) and energy (MJ DE or MJ NE) and the more comprehensive include ranges of essential amino acids and fatty acid residues, minerals, trace elements, vitamins, oil, fibre (crude, neutral detergent, dietary etc.) and ash. Certain of these characteristics provide limited nutritional information, but their declaration may be a statutory requirement on product labels within the EU (see Chapter 5). The computational technique is typically known as 'linear programming', as it is assumed that each additional amount of a nutrient contributed by one ingredient, substituting for another ingredient's nutrient, causes a similar quantitative response.

More complex, nonlinear models of feed formulation (e.g. integer and quadratic programming) are also accommodated in some computer packages, allowing, for example, for the calculation of a formula in whole units of the ingredient. The term 'least-cost' is used as the computation calculates a mix at minimum cost, consistent with formulation nutrient levels that are not less than the minimum requirements plus safety margins, that is, minimum nutrient constraints. The safety margins allow for variation in requirements of individual animals and deviations in ingredient composition from those assumed. Maximum and even minimum formulation levels for many ingredients are also set as ingredient constraints for reasons that are listed below.

The requirements of the formulation, expressed in terms of nutrient and ingredient constraints, take the form of a set of linear inequalities. For example, if $x_1, x_2 \dots x_n$ represents the amounts (g/100 g) of different ingredients in the mix with protein contents $a_1, a_2 \dots a_n$ (g/100 g), then the inequality:

$$a_1x_1 + a_2x_2 + \dots a_nx_n \geq b \quad (25)$$

expresses the requirement that the protein content of the feed should not be less than b . In addition to such constraints, a similar linear combination of x_i , with cost coefficients in place of the analytical values, represents the total cost to be minimized. The solution provides a set of x_i (the formulation) which satisfies the constraints at minimum cost.

Feed manufacturers maintain, and regularly update, a database of analytical and cost information on a wide range of potential ingredients together with a set of nutrient and ingredient maxima and minima for each type of feed. Least-cost formulations are then produced automatically from this information periodically, as required. The computation also produces other values that give guidance on the sensitivity of the formula to small changes in the constraints, or costs.

Graphic example of linear programming

(An alternative simplified form of ration formulation is given in Appendix A.) The linear programming method of computing a least-cost formulation can be illustrated graphically for three ingredients, as shown in Fig. 6.12, whereas the computer may handle 30 or more potential ingredients. The two axes at right angles represent the percentages of the first two ingredients (x_1 and x_2) in the diet. The percentage of the third ingredient (x_3) in the diet is then determined, since all three inclusion rates must sum to 100.

Each point within triangle (a) ABC represents a particular formulation, since the percentages of ingredients x_1 and x_2 determine the percentage of ingredient x_3 . Negative inclusion rates are impossible, so admissible diets are restricted to the region defined by the triangle ABC (the hatched area), where the line AB represents mixtures of 100% ingredient x_1 and 0% ingredient x_2 to 100% of x_2 and 0% of x_1 , excluding ingredient x_3 . Point C represents 100% of ingredient x_3 and at point D the mix would contain 50% each of ingredients x_1 and x_2 and 0% of ingredient x_3 . As indicated, a mix at point M_1 would contain 40% of ingredient x_3 and 50% and 10%, respectively, of each of the other two ingredients. Several of these mixes would be completely unacceptable; thus, ingredient and nutrient constraints are imposed. Reasons for such constraints include the following:

- A particular ingredient may contain an essential nutrient that is not accommodated in the programme and so the ingredient's inclusion at a predetermined minimum level may be necessary.
- Excessive amounts of some ingredients, e.g. molasses, may reduce horse acceptance of the product, or may cause a digestive upset. This upset can result from unacceptable concentrations of anti-nutritive substances, such as anti-proteases and glucosinolates (Chapter 5), or from excessive concentrations of some nutrients.
- Manufacturing rate and quality can be facilitated, for example, by the inclusion of minimum amounts of fat and pellet binders or exclusion of large amounts of abrasive ingredients.
- The provision of moderate amounts of each of several ingredients, rather than large quantities of one ingredient, is normally to be preferred. This policy should reduce the risk of problems arising when there are deviations in composition and quality of ingredients compared with the values attributed to the ingredient(s) in the programme.

Table 6.24 Assumed feed analytical values used to compare DE and NE systems in Table 6.23.

	DE (MJ/kg DM)	CP (g/kg)	UFC (per kg)	MADC (g/kg)
Hay	6.88	82	0.41	40
Barley	14.98	110	1.16	92
Soya-bean meal	14.73	499	1.06	437

the region PQRST. The restriction of barley to a maximum of 60% does not affect the outcome in this example.

Diets of equal cost lie along parallel lines. In Fig. 6.12 the costs assumed give a line with a negative slope of 0.5. By moving the line in the direction of the arrows, so reducing cost, we find the diet of least cost. This will lie on the boundary of the feasible region, normally at a vertex. If no ingredient constraints had been applied, the mix M_2 , containing 73% of ingredient x_2 and 27% of ingredient x_3 , would have been obtained. As a result of applying the four constraints, the least-cost mix is that of M_3 at point Q, with the proportions 20%, 60% and 20%, respectively, of ingredients x_1 , x_2 and x_3 .

Figure 6.12 also shows that, in this example, the critical constraints determining the position of the optimum are the lines PQ (minimum protein requirement) and RQ (minimum bran requirement), whereas the outcome would be unaffected by changing the position of the bran and protein maxima. Increasing the cost of beans to £250/1000 kg would reduce the slope of the cost line (make it more negative) to -0.8, and so change the optimum from M_3 to P. As the cost of beans increases past £200, the least-cost composition abruptly shifts from that at M_3 to that at point P. In practice, any such large change would be introduced in several small discrete steps, at intervals of time, to avoid possible implications regarding the health of the horse. The professional formulator will know the acceptable maximum size of each step for particular ingredient substitutions.

In conclusion, the practice of linear programming has improved the reliability and cost-effectiveness of commercial diets. It allows formulations to reflect economically the most recent established scientific evidence with minimum risk.

Procedure for calculating diets by the DE and NE systems

The sequence of calculations necessary in calculating a simple diet using the NRC DE and INRA NE systems is given in Table 6.25.

APPETITE

There is much yet to be learnt about factors that influence the appetite of horses, but reluctance or eagerness to eat can be assessed either as the amount of dry feed

Table 6.25 Procedure for calculating ingredient make-up of a simple ration using NRC and INRA systems.

Calculation	NRC DE system	INRA NE system
(1) Assess daily feed capacity of horse	kg/day, Fig. 6.4	kg/day, Fig. 6.4
(2) Assess daily energy requirement of horse	DE/day, Table 6.6	UFC/day, Table 6.15
(3) Divide (2) by (1)	DE/kg	UFC/kg
(4) Calculate proportions of roughage:cereal	Appendices A and C	Appendices A and C
(5) Assess protein requirements per kg feed or per day	Table 6.7, CP	Table 6.15, MADC*
(6) Assess Ca and P needs per kg feed (90% DM)	Table 6.7	Table 6.7
(7) Calculate protein, Ca and P of mix	Appendices A and C	Appendices A and C
(8) Calculate soya, Ca and P additions necessary	Appendix A, Table 6.7	Appendix A, Tables 6.7 and 3.2
(9) Estimate other mineral and vitamin needs and add	Table 6.21	Table 6.21

*For relationship of DCP to MADC see equation (22), and to obtain MADC/kg feed divide daily MADC requirement by feed capacity per day (note: DM and 90% of DM feed estimates).

consumed consistently per day, or the amounts consumed in single meals. The ingesta derived from very coarse, poorly digested, long-fibrous feeds, if present in significant amounts, will be retained longer in the large intestine and depress the daily intake of DM. Although this is generally a disadvantage, it can occasionally help to contain the appetite of overfat animals. The proclivity of a horse to start eating energy-yielding feeds also depends upon the relative absence of products of digestion, including glucose, in the small intestine. To a lesser extent, concentrations of blood glucose may play a small part.

Under conditions of natural grazing, individuals will feed during perhaps 15 or 20 periods throughout the day. A series of small meals reflects not only the low capacity of the stomach, but also, probably more directly, the switching-off mechanism of digestion products in the small intestine. High caecal concentrations of VFAs, especially of propionate, have an immediate but small depressing effect on appetite by extending the interval between meals in ponies fed *ad libitum* and by reducing meal size at the time, yet they have no sustained effect over 24 hours. Lower increases in caecal VFA may even stimulate appetite.

Although many factors influence the capacity for feed, an estimate of the daily appetite of average healthy animals for leafy hay and oats can nevertheless be given (see Fig. 6.4). In the stable, horses are fed generally by the bowl, and overfeeding, or apparent loss of appetite, frequently reflects lack of recognition on the part of the groom of differences in the bulkiness and energy density of feeds (see Table 5.5, p. 131). For example, taking both these characteristics into account, many of the better coarse feeds currently available provide 20% more energy per bowl than is provided by a similar volume of crushed oats. Failure on the part of the horse to eat up may then simply mean that it has been given 20% more energy in its hard feed than it is familiar with, or requires.

Feed in an unacceptable physical condition, and more importantly with an unfamiliar or unattractive aroma, will also be a deterrent to the initiation of feeding. Stale feed, which has incurred the oxidation of many of its less-stable organic components, is unlikely to encourage a horse to eat. Occasionally, spices are used to cover, or camouflage, inadequacies of general acceptability, but they are generally to be discouraged as horses can become 'hooked' on these additives.

There is often glib talk about the appetite of horses for particular feeds, but apparently they have a true appetite only for water, salt and sources of energy, and if given the choice would not select a balanced diet. Hence, it is necessary to induce horses to consume mixtures most appropriate to their needs. Thirst will sometimes drive a horse to consume poisonous succulent plants, as a source of moisture, in an otherwise arid landscape. The appetites for water and for salt are interrelated. Thirst depends to a considerable extent on dehydration, or increased osmotic pressure of body fluids. When blood is hypertonic, horses will normally drink; if much salt has been lost through sweating, the body may be dehydrated, but the fluids will be hypotonic and thirst is then engendered by giving salt. The appetite for salt varies among horses – deprivation causes a greater craving in some than in others, despite a more uniform essentiality. On the other hand, an extreme thirst probably takes precedence over appetites for both salt and energy.

RATE OF FEEDING

A too-rapid consumption of cereals and concentrates by stabled horses is sometimes a problem needing attention. Although the consumption of long hay is a relatively slow process, cereals and other concentrates are normally eaten first at a particular meal (there is a case to induce the consumption of some roughage first; see Chapter 2), but the form in which the concentrate is given can have some impact on the rate at which it is consumed. German workers (Meyer *et al.* 1975b) showed that 1 kg feed in the form of oat grain or a pelleted concentrate took horses weighing 450–550 kg about 10 min to consume, but meal took longer. Feeding time and the number of chewing movements were, however, increased by 3–100% if 10–20% of chopped roughage was added to the oats or concentrate. Finely ground meal mixes took longer to eat than crushed grains or pelleted feed; the addition of chopped roughage to fine mixes speeded up intake. Poor quality retarded consumption rate, suggesting a place for good quality barley straw in occupying the time of greedy horses.

Many horses develop vices, such as wood chewing, faeces eating (coprophagy) or, less frequently, cribbing (forced swallowing of gulps of air) (see Plate 11.1, p. 454). Recent evidence indicates that wood chewing and crib biting are responses to gastric inflammation and erosion. Such horses have a lower mean postprandial gastric pH with a wider pH range than is found normally (Lillie *et al.* 2003). The wider range may indicate variable biting, and so a variable salivary flow. The provision of an antacid supplement was shown to be associated with fewer gastric ulcers and a reduction in crib biting (Badnell-Waters *et al.* 2003). Boredom in isolated boxes is a

contributory factor in vice initiation and the provision of long hay or good-quality straw does help to circumvent this, but does not eliminate its occurrence (see also 'Ailments related to housing, Weaving', Chapter 11).

Sound teeth enable horses to grind roughage to a small particle size, but decaying teeth, or molars with sharp points abrading the cheek and tongue (see Fig. 1.1, p. 4), encourage bolting and poor mastication with the consequences of rapid intake, choking and sometimes colic. Dental treatment is clearly indicated, together with the provision of coarsely- and freshly-ground cereals and roughage chopped in short lengths. These may be given mixed together in a wet mash, which in turn may reduce wastage. The dampening of feed is, however, generally unnecessary except when large quantities of bran and beet pulp are given, in which case time for the thorough absorption of water should be allowed so that their true bulkiness is realized. Linseed also swells considerably on soaking and boiling; the latter, of course, is to be recommended, as described in Chapter 5. Where feeds are dusty, damping down lays the dust, so decreasing the likelihood of irritation to the respiratory tract in susceptible individuals. The damping of feed may also inhibit the segregation of its components. If feeds are too dry, minerals, for example, may sift to the bottom and be left unconsumed in the manger.

PROCESSING OF FEED

Preferred methods for processing raw cereal grains are coarse grinding, cracking, rolling, crimping (passing between corrugated rollers), steam flaking and micronizing. The overall objective of each is to improve digestibility and acceptability, but the last two may accelerate the onset of satiety during a meal. The processing of oats and barley is generally difficult to justify if the costs exceed 5% of the cost of the raw material, but it is necessary shortly before feeding for the small grains milo and wheat (see also discussion on laminitis, Chapter 2). Steam flaking or micronizing tends also to extend shelf life, destroys most heat-labile anti-nutritive substances and increases the proportion of starch digested in the small intestine when intake rates are high. Normal cooking is, however, inadequate for the destruction of some mould toxins found in poorly harvested crops (see also Chapter 5).

FEEDING FREQUENCY AND PUNCTUALITY

Horses, like most other animals, are creatures of habit, and their reactions are in part affected by an inner clock. Thus, the wise groom sticks to regular feeding times, week in and week out. With regular exercise, metabolic upsets and accidents can be avoided, and damage to stable doors through chewing and kicking can be decreased. Designs for feed mangers and water bowls recommended by the French Ministry of Agriculture are shown in Figs 6.13 and 6.14.

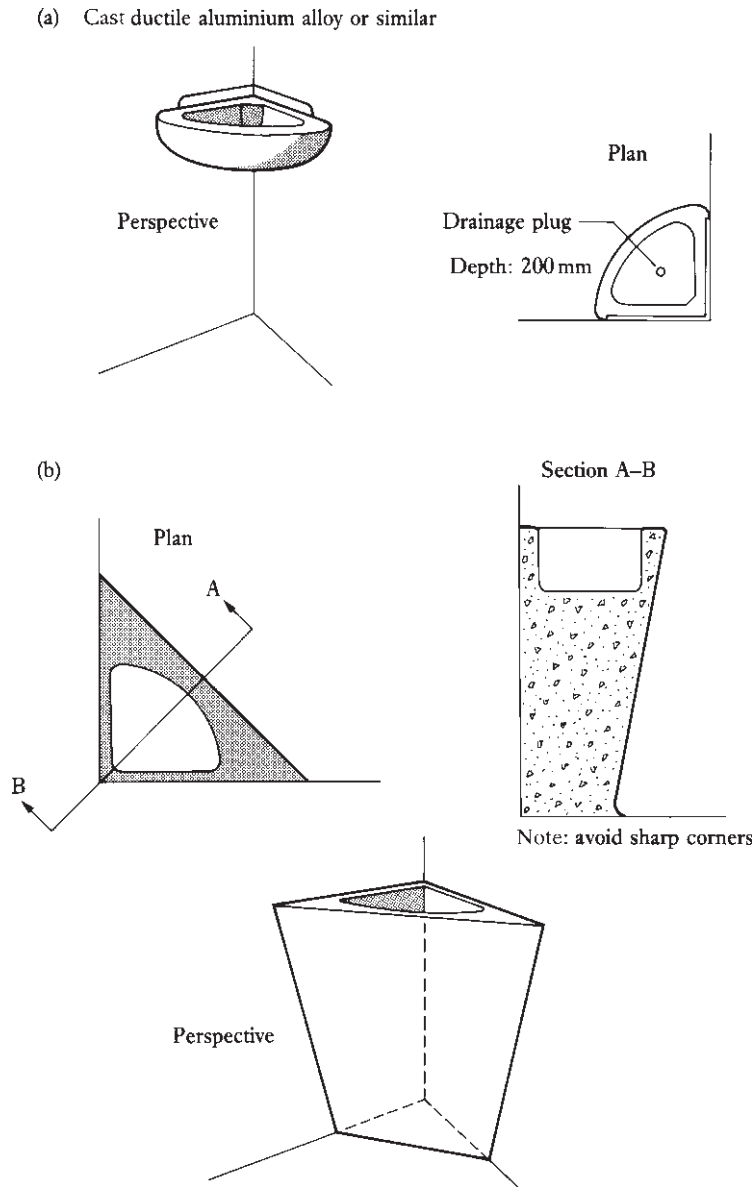


Fig. 6.13 Designs for feed mangers: (a) unbreakable manger; (b) manger embedded in concrete (Ministère de l'Agriculture 1980).

CEREAL COSTS AND THEIR ENERGY CONTENTS

Cereals, and more particularly oats, of low bushel weight provide less DE per kilogram than do cereals of higher bushel weights. Digestive upsets are minimized, when changing from one feed or cereal type to another, by ensuring that the DE, or

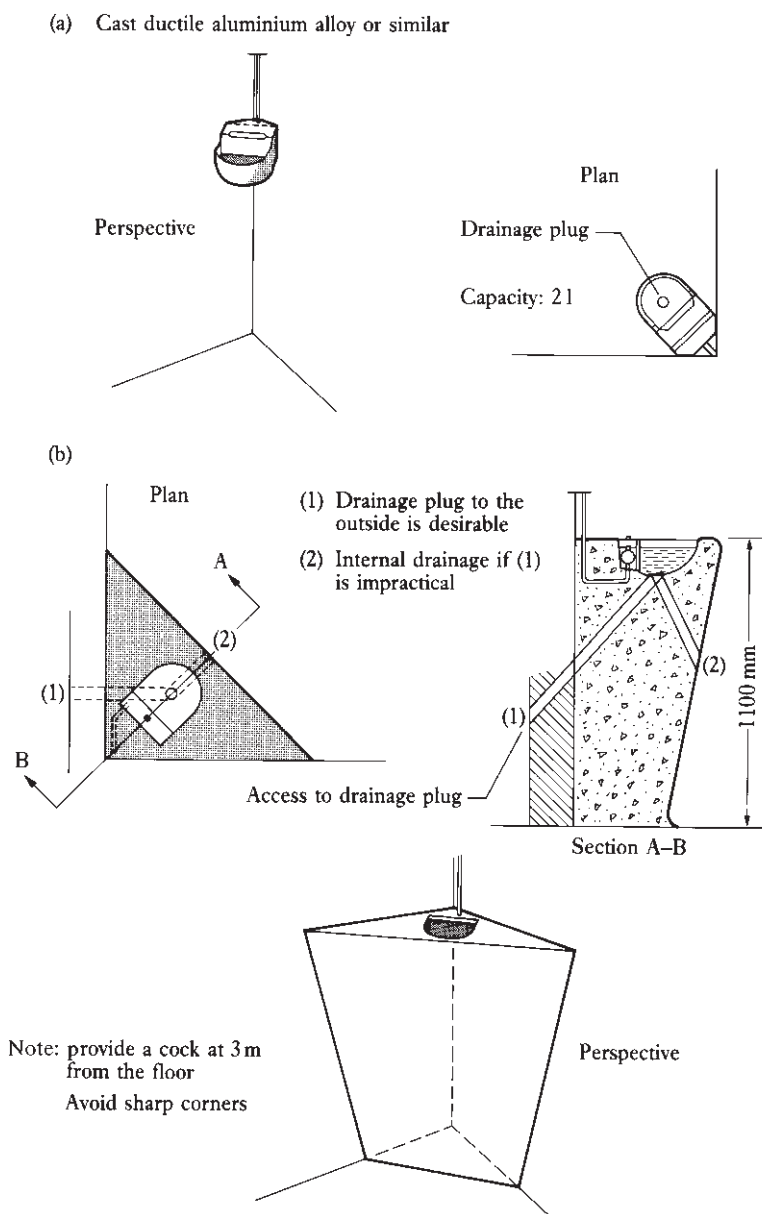


Fig. 6.14 Designs for water bowls: (a) unbreakable water bowl; (b) water bowl embedded in concrete (Ministère de l'Agriculture 1980).

NE, intake does not simultaneously rise. A bowl of an average sample of oats may provide only 56% of the DE of a similar bowl of average wheat (see Table 5.5, p. 131). As a result of its bulkiness, oats tend to be a safer feed than the other cereals although, through careful and expert feed management, no differences in this respect should arise. Therefore, in assessing the feeding value of the various cereals,

account should be taken of the purchase price per unit of DE or NE (see Appendix C).

TRADITIONAL FEEDS

In the past, many stables in the UK gave their horses a diet based on grass hay, oats and a little cooked linseed. Before the advent of the tractor-drawn baler, hay was probably made for horses with greater care than it is today, when the horse in many situations no longer reigns supreme in its claims to conserved forage and grazing privileges. In terms of nutrients, this diet now leaves a little to be desired, but the adult horse has some capacity to adapt to its culinary shortcomings, because it may take a considerable period of time for a large non-productive animal to be depleted of essential nutrients. Many of these nutrients are made good where summer grazing is available; nutrients provided by productive pasture in summer will serve as a reservoir for two months of the ensuing winter, but not generally until the spring grass again refurbishes losses. Lactation of the mare and early growth of young stock rely traditionally almost entirely on the availability of grazing, any deficiencies of which are soon revealed.

A diet of oats, hay and linseed is alien to the natural inclination of a browsing animal so that there is no good reason why other equally strange feeds should not be used if economies can be achieved and deficiencies rectified. There are now better protein sources than linseed and there are oils less prone to peroxidation than linseed oil, as these other oils contain much lower concentrations of linolenic acid and contain added antioxidants (but see discussion of n-3 vs. n-6 fatty acids in 'Polyunsaturated acids', Chapter 5). Stabilized forms of groundnut, soya, sunflower, rape and corn oils are widely available today. By understanding the necessity for protracted periods of adaptation, and by recognizing the differences in bulk and energy density among the various cereals, the careful feeder has a wide choice of feeds. The principal advantage of oats is to the novice feeder. This advantage could easily be supplanted (with a probable saving in costs per unit of energy) by the use of a variety of cereals and by-products, however, the traditional feeder knows by long experience how many bowls of oats a performance horse will consume without refusal. Without careful weighing, the quantities of alternative mixtures required can be misjudged and overfeeding or underfeeding can result.

COMPLETE DIETS

Many horses and ponies receive diets based on compounded nuts and long hay, or haylage. In normal circumstances this hay should be preferred to a source of ground fibre if the quality is good and the price acceptable. Compounded feeds described as complete pellets are for consumption by adult horses required to carry out moderate work where no other feeds are to be given. These pellets have the advantage of providing a reasonably balanced diet, relatively free from dust, for horses subject to

respiratory irritation and allergy. The absence of long fibre is likely to cause a higher incidence of vices, such as wood chewing and coprophagy, but these annoyances are undoubtedly preferable to the exacerbation of a respiratory problem. Rationing is simplified by the constant bulk and energy densities of the feed and storage space is minimized, but some problems of boredom require ingenuity to overcome.

SHELF-LIFE OF FEEDS AND FEED CONTAMINANTS

The grinding of cereals increases the surface area exposed to the atmosphere and deterioration proceeds continuously, retarded only by the presence of natural antioxidants. Staleness detracts from feed acceptability. Legislation permits the addition of limited amounts of synthetic antioxidants to products for consumption by animals. The most commonly used of these are butylated hydroxyanisole (BHA), butylated hydroxytoluene (BHT) and ethoxyquin (EU). Legislation permits 15 chemicals as antioxidants to be included in animal feeds (Schedule 3, UK Statutory Instruments 2000 No. 2481, The Feeding Stuffs Regulations 2000 and European Commission Regulation (EC) No. 2316/98; Anon 1995c). These substances are safe, have the effect of extending shelf-life and acceptability of feeds, and they cause no infringement of the Rules of Racing.

Some nutrients are particularly susceptible to destruction by light. Hence, concentrated feedstuffs should be covered during storage, as the products of light destruction are also harmful to the stability of certain other nutrients. Atmospheric oxygen brings about the gradual destruction of fat-soluble vitamins and unsaturated fats. Among the more critical water-soluble vitamins, thiamin (vitamin B₁) and pantothenic acid are somewhat unstable. The loss of these nutrients and other unstable compounds detracts from the value of feedstuffs. However, whole grains in an uncrushed or unground form remain relatively stable for several years with only a slight decline in feeding value, although this, of course, assumes good storage, freedom from pests, moisture levels of less than 135 g/kg (the lower the better) and absence of cracked and moulded grains. Discoloration of cereal grains may indicate only superficial microbial damage during ripening as observed when one compares good-quality Scots oats with their counterparts from Canadian or Australian sources. However, discoloration may be an indicator of more profound internal damage through fungal infection, which will seriously impair feeding value and stability in storage. Thus, bright and bold grains are to be preferred in the absence of other information. Further aspects of storage have already been discussed in Chapter 5.

Dressed seed corn or cereal grains that have been exposed to toxic pesticides should not be used for feeding to horses, although several pesticides leave quite harmless residues.

STUDY QUESTIONS

- (1) What is the sequence of actions and decisions to be taken on introducing a rationing system and meeting the nutrient requirements of a stud and a riding stable?
- (2) What are the merits and demerits of the two energy systems discussed: the NRC DE system and the INRA NE system?
- (3) What should be the characteristics of a satisfactory feed store?

FURTHER READING

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Chapter 7

Feeding the Breeding Mare, Foal and Stallion

You should wean your foals at the beginning of winter, when it beginneth to grow cold, that is about Martinmas, or the middle of November, and wean them three days before full moon, and hang about their necks upon a piece of rope seven or eight inches of the end of a cow's horn, to catch hold of them upon occasion, after which bring them all into your stable, with racks and mangers pretty low set.

S. de Solleysel 1711

THE OESTROUS CYCLE AND FERTILITY

The natural season for maximum breeding activity in both the mare and the stallion in the UK is from April to November, but the breeding season can be shifted by artificially changing daylight length and by manipulating the diet. A geographical move to the southern hemisphere can, of course, have comparable effects. During the season, the normal mare expresses consecutive oestrous cycles approximately 22 days long; within each cycle there is a period of oestrus of varying intensities that lasts on average six days. The fertility of the oestrus is low at the start of the season, but the creation of large follicles that ovulate and generate corpora lutea can be stimulated by the extension of day length and dietary adjustment, as the reproductive activity of both mares and stallions is under the influence of daylight length. A survey of 1393 TB mares visiting 22 stud farms in Newmarket, UK, revealed that mares were mated 1.88 times until diagnosed pregnant at 15 days after ovulation. Of the mated mares 89.7% remained pregnant by day 35 of pregnancy. Ultimately 82.7% of the mares surveyed gave birth to a live foal at term (Morris & Allen 2002).

Periods of darkness are associated with a rise in plasma concentrations of the hormone melatonin, much above that of daytime levels, so that, during the winter, melatonin is secreted for a greater length of time each day (Domingue *et al.* 1992). Ovarian activity and follicular growth occur during the late spring and summer months in response to increasing daylight length, reducing the nocturnal melatonin response, and acting on some endogenous biological rhythm. Sequential periods of 16 hours of light followed by 8 hours of dark are optimum for inducing ovarian activity. This seasonal pattern, modulated by light, can be advanced by about 21 days through stabling. Thus, an extension of daylight to 16 hours, an increase in the plane of nutrition and possibly a rise in ambient temperature during the cold months (December in the northern hemisphere) will stimulate the onset of normal cycling two to three weeks earlier in the first months of the year.

This situation is consistent with the observation that two-year-old Welsh Moun-

tain pony colts daily ate greater quantities of a complete pelleted diet when the daylight length was extended to 16 hours (220lux) cf. 8 hours (220lux) (Fuller *et al.* 1998, 2001). The cyclic changes in growth rate of the body and pelage were synchronous with those of feed intake. However, interestingly, these three related physiological responses were delayed by five to eight weeks following change in the day length.

It seems that cyclic hormonal responses to seasonal photoperiod occur in both sexes. Argo *et al.* (2001) determined that in prepubertal pony colts changes in pelage (coat), plasma prolactin, follicle-stimulating hormone (FSH) and testosterone concentrations were under the influence of photoperiodic mechanisms, but that luteinizing hormone (LH) release may be blocked by immaturity or active suppression.

High fertility tends to coincide naturally with the flush of grass in late spring. Successive oestrous periods will be of increasing fertility in healthy, barren or maiden mares, and individuals that are increasing in body weight are more likely to conceive. Therefore, by starting with a lean individual in November and December, this objective is more likely to be achieved. It has been suggested that forcing barren mares in December and January enhances the probability of twins, yet if early conception is desirable such a procedure is obligatory.

Energy intake is important for ovulation and embryonic development but less so for foetal growth (Meyer 1998). An excessive energy supply may favour twins, whereas restriction prior to parturition can induce premature birth. On the other hand, protein restriction seems to be important solely with extreme under-nutrition of the mare. Mares with body condition scores (BC) of only 3.0–3.5 (max. 10) during the anovulatory period (autumn and winter), experienced longer and deeper anoestrus than did those with good body condition. This response was accompanied by lower plasma leptin, insulin-like growth factor-I (IGF-I) (see Glossary for these terms) and prolactin concentrations (Gentry *et al.* 2002). Subsequent work by this group indicates that there is an interaction between leptin secretion and the secretion of the hormones insulin, T_3 and growth hormone (Cartmill *et al.* 2003). Thus, serum concentrations of leptin are positively correlated with condition score, but unrelated to level of feed intake (Buff *et al.* 2002).

Changing body condition score (BC) affects fertility. When BC was changed from 7 in September to either 8 or 3.0–3.5 in January, by feed control, Gentry *et al.* (2001), in Kentucky, found that a low score caused deep anoestrus, whereas the high-BC mares continued to cycle. A thyrotropin-releasing hormone (TRH) interacts with growth hormone (Pruett *et al.* 2003) and a TRH challenge causes a rise in circulating thyroid hormone in all horses. Powell *et al.* (2003), however, observed a prolactin response in geldings to TRH only in horses receiving large amounts of concentrates. Gentry *et al.* (2001) reported a higher prolactin response to TRH and higher luteinizing hormone (LH) response to gonadotropin-releasing hormone (GnRH) by mares in high condition, pointing to a mechanism for the effects on fertility.

The inclusion of animal fat, rich in cholesterol, to the diet of pregnant mares may also stimulate circulating levels of LH and fertility by providing a precursor of steroid hormones (Hagstrom *et al.* 1999). In addition, exogenous hormones of

various origins have been used widely to hasten ovulation (Hardy *et al.* 2003). Diets based on starch, compared with those rich in fat and fibre, should cause reduced insulin sensitivity in barren mares and increase the risk of overt insulin resistance (Hoffman *et al.* 2003a). Subsequently this group (Hoffman *et al.* 2003b) concluded that the maintenance of an ideal body condition in geldings, and the avoidance of meals rich in sugar and starch, would decrease the risk of insulin resistance. In contrast, for reproduction, where glucose demand and hormonal relationships are different, no difference in glycaemic effects have been noted between fibre and fat v. starch and sugar diets (Williams *et al.* 2001a, b).

Information relative to the potential control of reproductive cycles by infusion of amino acids was produced by Sticker *et al.* (2001), whence pituitary and pancreatic hormone release was influenced. The dicarboxylic amino acids, aspartic and glutamic acids, each given in the dose of 2.855 mmol/kg BW in water, caused the release of growth hormone; whereas the diamino-mono-carboxylic acids arginine and lysine, each at 2.855 mM/kg BW in water, caused the release of prolactin and insulin. The glutamate receptor agonist N-methyl-D,L-aspartic acid (1 mg/kg BW in water), stimulated growth hormone and gonadotropin release. Whether spring grass exerts a hormonal influence by this mechanism is as yet unclear. Nevertheless, it is evident that photoperiod and condition score influence the onset of cycling in barren mares, but the optimum composition and energy content of diet in mares of good condition have still to be established.

Freedom in the quantities of feed consumed seems possible where it is composed largely of hay. Doreau *et al.* (1990) offered pregnant mares of Anglo-Arab and Selle Français breeds *ad libitum* intakes of a 90:10 mixture of hay and concentrates from four weeks before foaling until five weeks after foaling. The hay was either of poor or high quality. The intakes for the poor and high quality mixtures before and after foaling averaged respectively 11.1 v. 12.4 kg DM and 18.6 v. 21.1 kg DM. The condition score of the mares receiving the lower quality diet was relatively poor at foaling and the consequential shortage of energy and inadequacy of protein (342 g, 426 g and 579 g MADC, see Chapter 6) during the last month of pregnancy, the first week of lactation and weeks two to five of lactation, respectively, led to poorer foal growth rates to five weeks of age without having influenced birth weight. Although the mares were initially in similar condition, the outcome may have partly depended upon the extent of energy reserves of the pregnant mare. If slightly lower foal growth rates are acceptable, adequate performance has been observed by Micol & Martin-Rosset (1995) under upland grazing conditions amongst French mares of heavy breeds (700–800 kg BW) such as Breton, Comtois and Ardennais, which may lose 17–25% of their body weight between foal weaning and the next foaling. Of this loss, 12–14% is products of conception and 5–10% loss of body mass, indicating the mares' reserves had been liberally incorporated in foal tissues. Nevertheless, pregnant mares should be kept fit but not fat as this reduces foaling difficulties and provides greater freedom for controlling milk secretion by feeding during lactation.

The foal heat occurs within 14 days of foaling and subsequent heats occur at 22-day intervals in unbred mares. The recommended rate of feeding of lactating mares

is given in Table 6.6, p. 206, yet it has not been clearly established whether this is the optimum rate for maximum fertility of the foal heat and subsequent oestrous periods. Milky mares, nevertheless, have a greater tendency to resorb fertilized eggs at first oestrus. This could be the reason for a putative association between overfeeding during the last three months of pregnancy and a reduced subsequent fertility.

Unfortunately the experimental evidence to support this assertion is conflicting. Jordan (1982) noted that no reduction occurred in conception rate among pony mares losing 20% of body weight during gestation, but allowed to gain weight during lactation. Heneke *et al.* (1981) reported that mares in this condition at foaling had reduced conception rates, longer postpartum intervals and more cycles per conception. Conception rates of mares in good condition at foaling, but who lost weight during lactation, were as good as those of mares in good or thin condition at foaling that maintained or gained weight in lactation. Evidence in the USA indicates that mares foaling in a fat condition should be allowed to hold their weight, rather than lose it, and that thin mares should gain weight during lactation in order to maximize the pregnancy rate at 90 days postfoaling. It is concluded that thin mares should be fed well in lactation to stimulate fertility.

Experience with dairy cows might suggest that if mares are fed too liberally through gestation and given inadequate feed during lactation they are more prone to a fatty liver condition, known to reduce fertility in the dairy cow. Observation of both horses and ponies shows that various stresses during late pregnancy and early lactation, accompanied by an inadequate and impoverished diet, predispose the mare to an extreme metabolic upset associated with loss of appetite, abnormal reactions, diarrhoea, hyperlipidaemia and eventual death. This represents a breakdown in energy metabolism with liver fat accumulation, as happens in the dairy cow. Any imposed weight reduction in obese pregnant mares should therefore take place before the last three months and, for preference, the fatness should be corrected before breeding is instigated. This may be achieved by providing the mare with good-quality hay, but no concentrated feed.

GESTATION

The gestation period of the mare commonly lasts for 335–345 days but may continue for one year. The period in part depends on the month of breeding. In the northern hemisphere, early-bred mares (that is, those conceiving before the end of April) normally have a gestation period exceeding 350 days and up to 365 days. Those bred in May normally foal after 340–360 days, and those bred in June and July generally foal after 320–350 days. The critical factor may be day length during the last three months of pregnancy, as when the photoperiod was artificially extended to 16 hours in late gestation of Quarter Horse mares (Hodge *et al.* 1981), the gestation period was shortened by 11 days and the interval from parturition to first ovulation was decreased by 1.6 days in comparison with mares subjected to natural day length.

Where diets are grossly imbalanced in terms of protein and minerals, especially Ca and P, the foal will be adversely affected at birth and reduced milk yield and infertility will ensue. A marginal Ca intake retards foetal growth and iodine and selenium status have considerable effects on fertility of the mare and the viability of the foal (Meyer 1998). A deficiency or excess of either iodine or selenium depresses embryonic development and viability of the foal (the author's observations and see 'Trace elements', Chapter 3). An extreme and chronic deficiency of vitamin A may prejudice embryonic development and the ovarian cycle (Meyer 1998), whereas reports on specific effects of β -carotene have been contradictory.

Protein and energy requirements

(See Chapter 6 for feed requirements of the pregnant mare). Definitive statements on protein and energy requirements for breeding mares are as yet not possible for two reasons:

- (1) A large well-fed mare has considerable reserves of energy and protein on which she can draw during pregnancy if daily intakes fall below recommended levels.
- (2) A reduction in intake generally seems to induce economies in metabolism so that deficiencies are partly offset.

Clearly the mare is capable of considerable adjustment to a variety of situations. However, in extremes, excesses or deficiencies of energy will lessen her reproductive efficiency. During winter or summer the ribs of a mare should not be seen, but should be detectable by touch with no appreciable layer of fat occurring between them and the skin. The condition of the over-fat mare can be improved by gradually reducing the cereal component of the ration while the protein and mineral mixture is maintained at the previously determined level of intake. Meyer (1983b), in Hanover, concluded that the mare should be 18% above normal weight before parturition to achieve a high fertility after parturition.

Pregnant mares are normally kept on pasture. Australian workers (Gallagher & McMeniman 1988) established that grass/legume pastures in southeast Queensland could support the nutritional requirements of TB brood mares by providing DE intakes of 68.0 and 91.7 MJ/day (the latter figure is approximately 10% higher than the NRC 1989 recommendation) and digestible N intakes of 91.2 and 138 g/day during mid and late pregnancy, respectively.

During periods of inadequate energy intake epinephrine secretion increases fat mobilization from body reserves leading to a rise in plasma NEFA concentrations in blood. In the normally fed human subject receiving three meals daily, plasma NEFA concentration decreases after each meal and rises before the next meal. In the horse, plasma NEFA begins, and continues, to rise from four hours following an afternoon meal in adult mares given 50% of their protein and energy needs for maintenance in two daily meals, and not in those given 100% of those needs (Sticker *et al.* 1995). Thus, this measurement may provide a useful means of assessing energy status of the horse.

PARTURITION

In the 24 hours before birth of the foal, the mare should be fed lightly with good-quality hay and a low-energy cereal mixture including bran, or proprietary horse and pony nuts (10–11% protein, 3% oil, 14–15% crude fibre; see Table 7.1), with access to restricted quantities of warm water. The first feed after parturition can effectively be a bran mash and the second can include some bran with small quantities of good-quality proprietary stud nuts (16–17% protein, 3% oil, 8% crude fibre; Table 7.1) or a cereal protein mixture. Obese mares tend to be less active and so poorer muscle tone may lead to birth difficulties and delayed expulsion of the placenta, which should be passed during the first hour after birth. The rate of concentrate feeding up to day ten should be restricted in order to avoid excessive milk secretion and digestive disturbance in the foal. However, inadequate amounts of energy may contribute to the metabolic abnormalities outlined in ‘The oestrous cycle and fertility’, this chapter. Recommended allowances are given in Table 6.6.

Perhaps 5% (Rossdale & Ricketts 1980) or 10% (Jeffcott *et al.* 1982c) of foals may be lost through perinatal mortality, including stillbirths and postnatal deaths. Of these, significantly more are male. Although nutrition is a vital factor, the significance of this statistic is entirely unknown. Birth weight is a crucial factor in determining the prospects of foals and, despite the influence nutrition can have on this,

Table 7.1 Composition of foal milk replacer, stud concentrate mixture and horse and pony mix to be given with hay and water.

Foal milk replacer (see footnote for mixing ¹)	(%)		Stud mixture ² (%)	Horse and pony mix (%)
Glucose	20.0	Oatfeed	—	25.0
Fat-filled powder (20% fat)	5.0	Oats	46.0	33.0
		Wheat bran	15.0	20.0
Spray-dried skimmed-milk powder	40.0	High-protein grass-meal (16% crude protein)	15.0	10.0
Spray-dried whey powder	32.7	Extracted soya-bean meal	15.0	4.0
High-grade fat ³	1.0	Molasses	5.0	5.0
Dicalcium phosphate	1.0	Fat ³	1.0	1.0
Sodium chloride	0.2	Limestone	1.0	0.9
Vitamins/trace elements ⁴	0.1	Dicalcium phosphate	1.1	0.5
		Salt	0.75	0.5
		Vitamins/trace elements ⁴	0.1	0.1
Total	100.0		100.0	100.0

¹Disperse in clean water at the rate of 175 g/l (for 2 days after colostrum at 250 g/l). Also can be pelleted and mixed with stud mixture as a weaning feed for orphan foals.

²This mixture is satisfactory as a creep feed and postweaning diet. However, a mix specifically for young weaned foals to be fed with grass hay could to advantage contain an extra 5% soya-bean meal replacing 5% oats.

³High-quality tallow and lard, including dispersing agent. Stabilized vegetable oil could alternatively be added at time of mixing.

⁴To provide vitamins A, D₃, E, K₃, riboflavin, thiamin, nicotinic acid, pantothenic acid, folic acid, cyanocobalamin, iron, copper, cobalt, manganese, zinc, iodine and selenium.

size of the dam is a major controlling influence. So, an acceptable minimum weight must depend on the breed and the purpose intended for the individual offspring. In TBs, an early rapid growth rate is normally expected and required for work at an early age. For this, foals of less than 35 kg probably should not be kept. Where twins are born, their total weight approximates that of large single births with a mean in the region of 55 kg for TBs, implying that it is practical to retain only the heavier of the two.

ACQUIRED IMMUNITY IN THE FOAL

The mare must pass adequate passive protection to her foal through the colostrum, and she therefore should have been situated in the foaling area for, at the very least, two and preferably four weeks before foaling. This means she will confer some immunity to the strains of microorganism peculiar to her environment, for example, those causing scours, joint ill and septicaemia. Newborn foals will normally first suck within 30–180 min of birth. Colostrum is rich in protein (particularly immunoglobulins), dry matter and vitamin A. If foals are deprived of colostrum, an injection of about 300 000 iu vitamin A is in order. Immunoglobulins do not pass through the dam's placenta and can be absorbed efficiently through the intestinal wall of the foal only during the first 12–24 hours of life. The major causes of colostrum deprivation in the foal are premature birth and delayed suckling, small-intestinal malabsorption, premature leakage of milk through the teats or death of the mare. The immunoglobulins are concentrated by the mare in her udder within the last two weeks of gestation, when their level in the mare's serum falls. There is, therefore, a selective concentration of this protein fraction in the mammary gland.

If the foal is suckled normally, the concentration of the immunoglobulin fraction in the colostrum 12–15 hours after birth is only 10–20% of the initial value. It is known that the protein content of mare's colostrum is around 19% during the first 30 min after parturition, but by 12 hours the level falls to about 3.8% and after eight days it reaches a fairly constant level of 2.2% (Ullrey *et al.* 1966). The foal absorbs γ -globulin as intact undegraded molecules throughout the first 12 hours of life. At birth the foal is agammaglobulinaemic but it responds to the colostrum, its serum γ -globulin rising for 12 hours to 8 g/l serum (Jeffcott 1974b–d). Amounts of specific antibodies so acquired by the foal's blood decline from 24 hours of age; by three weeks the values are halved and by four months the titre of antibodies provided by the mother is barely detectable.

The foal's own system for building active immunity in the form of autogenous γ -globulins first provides detectable products at two weeks of age in the blood of colostrum-deprived foals and at four weeks in those reared normally. By four months of age the γ -globulins have attained adult plasma concentrations. Up to this age, therefore, the foal is more susceptible to infection than is an adult in the same environment, particularly when it has received an inadequate quantity of colostrum, or colostrum at the wrong time.

If the mare has ejected much of her colostrum before foaling, then it will be necessary to give the foal colostrum from another mare, preferably one accustomed to the same environment or, failing this, cow's colostrum, rather than milk. Commercial sources of cow's colostrum are now available (see 'Bovine colostrum', this chapter). After 18 hours, the colostrum has little systemic immune value, although it does have some beneficial local effects within the intestinal tract.

A simple field test has been developed in which the turbidity of plasma is assessed following the addition of zinc sulphate; the results correlate well with concentrations of blood globulin in foals indicating whether sufficient antibodies have been absorbed in the neonatal period. This subject is discussed further under the section on orphan foals ('Blood plasma by parenteral or oral dosing', this chapter).

NEONATAL PROBLEMS

Hygiene is generally outside the scope of this book, yet the importance of cleanliness in the foaling area cannot be overemphasized. It is essential that the foal receives colostrum to provide it with some protection (passive immunity) from potentially harmful organisms in the environment. Nevertheless, the consumption of excessive quantities of milk can overload the digestive capacity of the foal and the milk may then become a substrate for rapid bacterial growth in the intestines. This situation can precipitate diarrhoea despite the consumption by the foal of normal quantities of colostrum.

The aetiology and treatment of foal diarrhoea involves several factors (Urquhart 1981). That caused by excessive milk intake and lactose intolerance is treated by decreasing or terminating milk consumption. The onset of so-called 'foal-heat diarrhoea' occurs 7–10 days post partum, frequently coinciding with the mare's first oestrus, but no correlation has been established between this form of diarrhoea and total solids, fat, protein or ash concentrations, bacterial count or oestrogenic activity of mares' milk. There are two problems concerning the feeding of the foal unrelated to microbial disease, haemolytic icterus and passing the meconium.

Haemolytic icterus

The foal's blood differs immunologically from that of its dam and on rare occasions the foetus may react with the dam's immune system, causing the production by the mare of isoantibodies to the foal's red cells. These antibodies are transmitted to the colostrum and the suckled foal may absorb sufficient to initiate a considerable destruction of red cells, precipitating an anaemia and jaundice, known as *haemolytic icterus*. In severe cases, the foal's urine will be discoloured with haemoglobin. If a mild attack is detected before icterus has occurred, the foal should not be allowed to nurse from its dam for 36 hours. Where the mare has previously produced foals with the condition, she may still carry similar antibodies. In this case the foal should automatically be given colostrum from another mare at the rate of 500ml every

1–2 hours for three to four feeds, followed by milk replacer until 36 hours, when it may be returned to its dam. In the meantime, the mare should be milked out by hand.

If the problem is anticipated, blood samples can be taken from the foal and the red cells allowed to settle. The abnormality causes red cell haemolysis (pink plasma rather than the normal straw colour). In these circumstances the foal may be severely anaemic and red cells from the mother may be slowly infused into a vein after removing the plasma and suspending the red cells in physiological saline. For preference, however, the source of red cells should be three or four geldings that have not previously received transfusions so the risk of immunological reactions is minimized.

A simple procedure has been proposed as a means of precluding damage *before* colostrum is taken by foals that are considered to be at risk. One drop of umbilical blood is mixed with four drops of saline and five drops of mare's colostrum on a clean microscope slide, checking after several minutes for agglutination reactions.

Passing the meconium

At birth, much of the large intestine, including the caecum and rectum, contains a substance, the meconium, which is normally completely voided within the first two to three days of life. Suckling usually sets up a reflex promoting defaecation of this material. If this does not occur, the normal passage of colostrum and milk may become blocked so that the gases formed during their fermentation cause distension and pain to the foal. It may then go off suck, act abnormally, crouch, lift its tail and flex its hocks in an effort to pass the offending material, or roll over in pain. Eventually a yellowish milk dung reaches the rectum, the meconium is cleared and the symptoms subside.

The problem is treated conservatively by administration of a lubricant through a stomach tube, plus one or two enemas of soap and water, or liquid paraffin, and the injection of pain-relieving drugs. If the foal goes off suck for an extended period it should be given a fluid feed by stomach tube or appropriate intravenous solutions. Intravenous feeding of glucose and an isotonic electrolyte solution (see Table 9.2, p. 325) is a life-saving procedure in cases of severe enteritis with consequent dehydration. In normal circumstances the foal will eat a quantity of its mare's faeces. In so doing it introduces beneficial microorganisms into the intestinal tract, which compete with pathogens present in the general environment.

LACTATION

At any given stage of lactation, the composition of mare's milk is remarkably similar among the various breeds of horse. The composition changes rapidly during the first days of lactation and then more slowly (Figs 7.1–7.4 and Tables 7.2 and 7.3). Milk contains about 2MJ gross energy/kg. Eight TB and two Standardbred mares

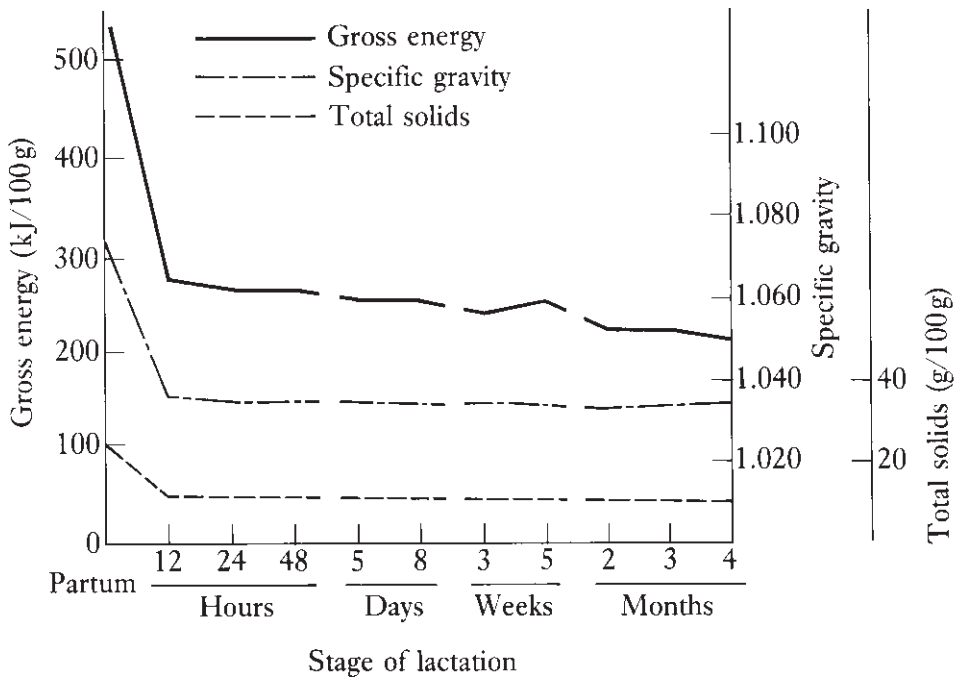


Fig. 7.1 Changes in specific gravity and concentration of gross energy and total solids in mare's milk at various stages of lactation (after Ullrey *et al.* 1966).

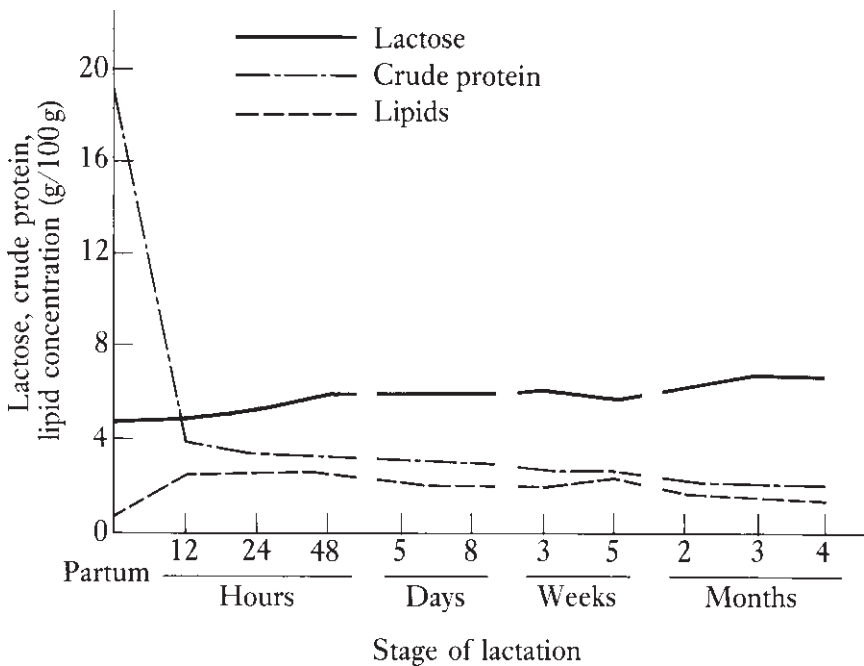


Fig. 7.2 Changes in concentration of lactose, crude protein and lipids in mare's milk at various stages of lactation (Ullrey *et al.* 1966).

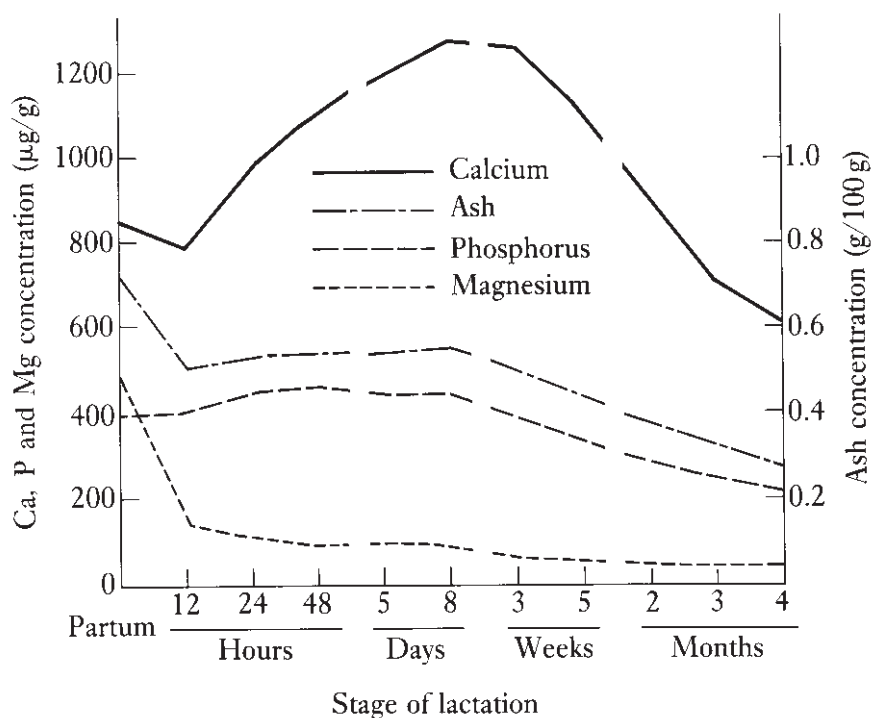


Fig. 7.3 Changes in concentration of ash, calcium, phosphorus and magnesium in mare's milk at various stages of lactation (Ullrey *et al.* 1966).

Table 7.2 Nutrient content of milk in Quarter Horse, TB, Dutch Warmblooded Saddlebred horse mares and some other breeds (Bouwman & van der Schee 1978; Gibbs *et al.* 1982; Oftedal *et al.* 1983; Schryver *et al.* 1986; Doreau *et al.* 1988; Saastamoinen *et al.* 1990; Martin *et al.* 1991).

	Dutch and some other breeds			Quarter Horse	TB
	Initial	28 days	196 days	2–150 days	24–54 days
Total solids (g/kg)	130	100–112	105	105	105
Gross energy (MJ/kg)	2.5	1.9–2.3	1.8–2	—	—
Fat (g/kg)	27	11–13	7	13–12	13
Protein (g/kg)	33	17–20	18	21–16	19
Ash (g/kg)	5.3	2	2.8	3	4
Calcium (g/kg)	1.2	0.7	0.8	0.8–0.6	0.9
Phosphorus (g/kg)	1	0.4	0.5	0.36–0.21	0.6
Lactose (g/kg)	58	66	66	—	69
Magnesium (mg/kg)	—	90	45	65–43	68
Potassium (mg/kg)	—	700	400	580–370	120
Sodium (mg/kg)	—	225	150	190–160	180
Copper (µg/kg)	—	450	200	—	200
Zinc (µg/kg)	—	2500	1800	—	2000

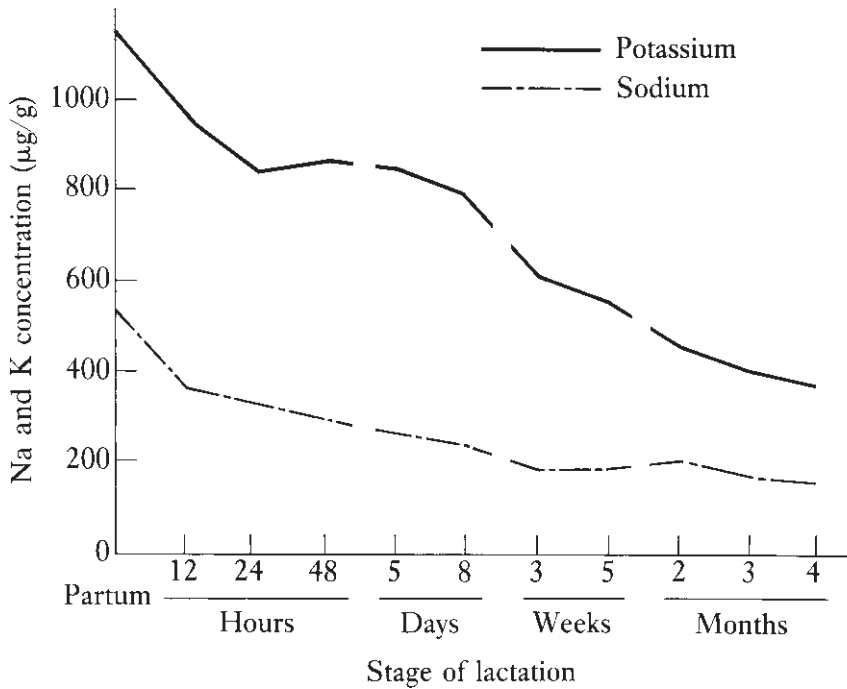


Fig. 7.4 Changes in concentration of potassium and sodium in mare's milk at various stages of lactation (Ullrey *et al.* 1966).

Table 7.3 The composition of mare's milk: amino acids* g/kg milk (Wickens *et al.* 2002).

	g/kg milk		g/kg milk
Arginine	1.17	Methionine	0.54
Histidine	0.56	Phenylalanine	0.90
Isoleucine	1.33	Threonine	1.22
Leucine	2.50	Valine	1.65
Lysine	1.70		

*These are considered to be the indispensable dietary amino acids, excepting the omission of tryptophan.

receiving a diet of concentrates and hay showed that they achieved yields of 16, 15 and 18 kg daily (3.1, 2.9 and 3.4% of body weight daily, or 149, 139 and 163 g/kg BW^{0.75}) at 11, 25 and 39 days postpartum, respectively (Ofstedal *et al.* 1983). Doreau *et al.* (1991) reported that, during weeks 0–5 of lactation, primiparous French Anglo-Arab mares, weighing 522 kg after foaling, produced less milk and of a lower fat content than was produced by multiparous mares (14.6 v. 16.6 kg/day and 16.5 v. 20.2 g/kg respectively). This difference was associated with a lower dry matter intake during pregnancy and lower plasma NEFA concentration during pregnancy and lactation for the primiparous mares. French draft (Breton, Comtois) mares,

Table 7.4 Typical milk yields (kg/day) by mares of various body weights during the 1st to 25th weeks of lactation.

Weeks	1–2	4–5	5–12	20–25
Quarter Horse (500 kg)	10	14	10	—
TB, Standardbred (494 kg)	12–16	14–16	18	—
Dutch Saddlebred horse (600 kg)	14	16	19	11
French draft (726 kg)	20	25	27	—

Table 7.5 Water requirements in the stud.

(a) Water provided by pasture herbage (kg) per kg herbage DM and total (including herbage) minimum water needs (kg) per kg feed DM consumed.			
Water content of herbage per unit DM		Water requirement of horses per unit DM consumed	
Spring growth	4	Last 90 days of gestation	3
Dry summer	2.5*	First 3 months of lactation	4
Mild winter	3	Breeding stallion	3
		Weaned foal	3
		Barren mare	2
*When 'burnt' and bleached the proportion of water may be as low as 0.15–0.3.			
(b) Minimum supplementary water requirements (l) per mare daily, assuming average milk yields.			
Live weight of mare: (kg)	200	400	500
Last 90 days of gestation: ¹			
in stable	12.7	22.3	26.4
on pasture ²	0	0	0
First 3 months of lactation:			
in stable	27.7	41.8	49.6
on pasture ²	7.3	10.9	12.3

¹The breeding stallion's needs are similar to those of the pregnant mare.

²These amounts will be insufficient for mares on parched pasture in environmental temperatures exceeding 30°C, where shade should be provided.

weighing 726 kg, yielded 20 kg/day in the first week, rising to 27.5 kg/day by week 8 (Doreau *et al.* 1992) (Table 7.4).

Milk yields are markedly influenced by the mare's innate ability, by feed consumption during the latter stages of pregnancy, and, more importantly, by water availability (Table 7.5) and intake of energy and nutrients during lactation. Experimental work with Quarter Horse and TB mares has shown that a reduction in the energy intake to 75% of that recommended for lactation by the NRC (1978) does not lead to a parallel decrease in foal weight at 75 days (Banach & Evans 1981a,b). Undoubtedly, the thriving mare has considerable capacity to adapt, within limits, to a restricted diet.

Mares weighing 400–550 kg may be expected to yield 5–15 kg milk daily during the first few weeks, 10–20 kg daily in the second and third months, falling to 5–10 kg by the fifth month; but normal yields assume an ample supply of water. Observations with Dutch Saddlebred horses indicated that suckling frequency averaged 103 times per day in the first week, falling to 35 times per day by week ten, and on each occasion the foal suckled for 1.3–1.7 min. (Small amounts taken frequently are unlikely to cause digestive disturbances.) At birth the foals weighed 57 kg on average and the weight doubled in the first 37 days of life.

Suckling TB and Standardbred foals have been shown to take in daily milk dry matter equivalent to 3.1%, 2.1% and 2% of body weight at 11, 25 and 39 days postpartum, when weight gain averaged 1.14 kg daily (Ofstedal *et al.* 1983). The comparable daily intakes of gross energy were 39, 32 and 37 MJ, respectively.

Effects of dietary energy, protein and urea on milk yield

Energy intake can be voluntarily modified to affect milk yield and composition. The voluntary food intake of draft broodmares was less when equal quantities of tall fescue hay and concentrates were given, compared with 95% hay and 5% concentrates (Doreau *et al.* 1992), causing differences in the protein and fat contents and in the fatty-acid profiles of the milk (Table 7.6). The milk from the high concentrate diet was richer in C18:2(n-6) (linoleic) and poorer in C18:3(n-3) (α -linolenic) acid. Thus, increased fibre and inadequate energy contents of feed may increase the fat and the protein contents of milk, but decrease yield. The effect on fat is similar to that in ruminants.

Dietary protein quality is known to affect the protein content of mare's milk. Glade & Luba (1987b) gave lactating mares 1.55 kg moderate quality protein/500 kg BW daily, or the same amount but with half provided as soya protein. This increased the protein content of the milk at seven days from 25.3 to 33.2 g/l. A difference persisted until the fourth week of lactation. Plasma methionine and lysine were in higher concentrations in foals nursing the soya-supplemented mares and their

Table 7.6 Effect of proportions of hay to concentrates in French draft mares (726 kg) on lactation (Doreau *et al.* 1992).

	Hay:concentrates 95:5	Hay:concentrates 50:50
DE (MJ/kg diet)	9.3	12.9
DCP (g/kg diet)	129	142
NE (UFC)*	0.65	0.89
MADC (g/kg DM)	74	100
Feed intake (kg DM/day)	22.9	21.4
Milk yield, week 4 (kg/day)	23.4	26.4
Fat, mean weeks 2 and 4 (g/kg milk)	14.7	11.6
CP, mean weeks 2 and 4 (g/kg milk)	26.3	24.4

*See Chapter 6, French INRA system.

Table 7.7 Mineral composition of mare's milk – electrolytes (mmol/l) and Cu and Zn ($\mu\text{mol/l}$) (Schryver *et al.* 1986).

Weeks postpartum	Ca	P	Mg	K	Na	Cu	Zn
1–2	33.3	29.2	4.64	17.0	9.4	12.1	44.4
3–4	27.9	22.6	3.66	13.0	7.5	7.5	35.2
5–6	23.1	19.5	2.96	10.0	8.0	6.8	30.6
7–8	21.6	19.0	2.47	11.5	6.5	5.0	29.1
9–14	20.7	18.2	2.18	9.8	6.4	2.7	27.5

withers height at seven weeks was greater. Poor pasture would therefore benefit from supplements of both good-quality protein and starch. Urea supplementation of mares given low-protein diets increases plasma and milk urea concentrations and reduces feed intake with adverse effects on milk intake and growth of their foals (Martin *et al.* 1991). Dietary energy source and amino acid intake within normal limits influence milk yield and composition and foal performance, but the evidence is insufficient for the recommendation of changes to existing requirement values.

Mineral composition of milk

The mineral composition of TB mare's milk was measured by Schryver *et al.* (1986) (Table 7.7) and other values are given in Table 7.2. The dry-matter content was found to decline sharply from 12% in the first week to 10.8% in the third, and then slowly to 10.2% by the 15–17th weeks of lactation in the study of Schryver and colleagues and as shown in Table 7.2.

Parturient paresis

Some high-yielding dairy cows suffer from a condition known as milk fever, or parturient paresis, which is probably caused by a sudden draining of blood calcium into the milk after parturition without an equivalent mobilization of bone calcium. A successful treatment entails giving cows a low-calcium diet two to four weeks before parturition and then providing a diet relatively rich in calcium during lactation. A regulated but high dose of vitamin D given eight to ten days before parturition has also been beneficial in some instances, but the dose has to be carefully calculated to avoid a gross and toxic excess. Pony and horse mares with a history of tetany (cf. paresis in ruminants) associated with depressed blood calcium in lactation might well benefit from being given a diet containing 1.5–2 g Ca/kg and approximately 3000 iu vitamin D/kg throughout the last two weeks of gestation. It is difficult to predict parturition with sufficient accuracy ten days before foaling and therefore single doses of large amounts of vitamin D at that time cannot be recommended. The diet should, of course, be adequate in other respects and during lactation the total diet of such mares should contain 5–6 g Ca/kg. Horses that are suffering from

this form of tetany are slowly given calcium gluconate intravenously while cardiac action is continuously monitored.

Grazing and drinking behaviour in the postnatal period

Grazing, lactating Welsh pony mares were shown (Crowell-Davis *et al.* 1985) to spend about 70% of their time feeding, whereas, excluding nursing, their foals at one week of age spent 10%, rising to about 50%, of their time grazing at 21 weeks. Foals tended to eat when their mothers were feeding, which was of greater intensity early in the morning and the evening. Generally, the entire herd moved to water sources together, and the frequency of this increased with increasing environmental temperature to 35°C. However, foals under three weeks of age did not drink, and half the foals were not observed to drink before weaning (also see 'Water requirements', Chapter 4).

Growth rate of the suckling foal reflects the rate of milk secretion of its dam. Among environmental influences on milk yield, the quality of pasture and free water availability are major influences. The supplementary feeding of lactating mares, which takes place in the stable at night or on the pasture, is another influence. Safe water sources are essential for lactating mares in hot weather.

Blood biochemical values of the foal

Normal blood characteristics of the foal are given in many texts, and the subject is referred to in Chapter 12. The serum values given in Table 7.8 indicate that the neonatal foal is deficient in globulins, and this must be rectified by consumption of colostrum before any other feed. Serum alkaline phosphatase activity decreases with age, probably reflecting the relative decrease in the rate of bone growth with increasing age, or emphasizing that mishaps in proper bone development may be established before or shortly after birth. Blood glucose and fat are in low concentrations at birth, reflecting the low energy reserves of the neonatal foal and its inability to maintain body warmth in a cold environment unless colostrum and milk are provided.

Table 7.8 Protein (g/l), neutral fat (g/l) and glucose (mmol/l) concentrations and alkaline phosphatase (ALP) activity (King Armstrong Unit) in the blood serum of TB foals (Sato *et al.* 1978).

Days from birth	Total protein	Albumin	Globulins	Neutral fat	Glucose	ALP
0	45.8	26.3	15.3	0.36	3.07	100
10	54.8	26.5	27.6	1.18	8.04	76
20	56.2	25.4	29.6	1.04	7.51	57
50	53.6	24.3	29.2	0.62	7.16	62
90	56.0	26.3	30.0	0.90	6.06	50
120	60.8	31.1	29.4	0.45	6.08	45

Creep feeding of the foal

Foals will start to nibble hay and concentrates at 10–21 days of age. If the milk supply of the dam, or the amount of grass, is inadequate, the provision of creep from this time may well enable a normal growth rate to be achieved. However, the principal objective of creep feeding is to accelerate the anatomical and physiological maturation of the GI tract of the foal so that, ultimately, weaning presents no particular stress or hazard and digestive disturbances caused by abnormal fermentation of ingesta are prevented.

Where the mare secretes minimal quantities of milk, a creep feed based on dried skimmed milk provided from about two weeks of age is recommended. The composition of this feed should be changed gradually to that of a stud nut (16–17% protein, 3% oil, 8% fibre) or a growing foal diet (17–18% protein, 3% oil, 7% fibre) (see Tables 7.1 and 8.6, p. 291) from 10–14 weeks of age. The use of milk pellets as a creep feed before weaning is contraindicated as it defeats the prime objective.

Control of growth abnormalities

Foals that are growing well with mares on pasture have no particular need for additional dry feed until two months before weaning, say at two to three months of age. Here the functions are to compensate for a waning milk production in the dam, to redress the effects of a decline in pasture quality and, probably more importantly, to accustom the foal to the dietary regime it must expect after weaning. Thus, the feed should be a concentrate of the type given in Tables 7.1 and 8.6 (p. 291) on which the foal will be maintained throughout the forthcoming winter and spring.

Supplementary creep feeds should be restricted in quantity to 0.5–0.75 kg/100 kg BW. Such a restriction will give a measure of control over the incidence of growth-associated ailments including epiphysitis and contracted tendons (flexural deformity) (Plate 7.1a–b). Where there is evidence of either of these, a restriction of growth rate, imposed by cutting the supplementary feed and by reducing the mare's feed for a period of three to four weeks, should not prejudice ultimate mature size if carefully regulated. The extent of restriction must depend on how serious the problem is. Foals that stand high on their toes at birth should be exercised regularly and allowed to grow at a submaximum rate if the condition is to be contained. Overtly contracted tendons at birth are relatively untreatable and possibly result from intrauterine malpositioning. If therapy is possible, splints or extension shoes are used and the foals are exercised regularly, with restricted access to feed until the abnormality is satisfactory (see also Chapter 8).

The vertical growth of normal foals is very rapid throughout the first 3–4 months of life. Access to creep feeders should be controlled by regulating the width of the entrance rather than by restricting its height. Foals of breeds with mature weights of 550 kg can be weaned easily when consuming nearly 1 kg of creep feed and 0.5 kg hay (or equivalent grass) daily by which time the foal should be in excess of 140 kg. These restrictions normally ensure that growth will not falter during the first



(a)

Plate 7.1 Chronic contracted tendons in a yearling showing enlarged fetlock joints and upright stance. Later improvement was achieved by desmotomy of the superior check ligaments. Before surgery (a) and after surgery (b).



(b)

Plate 7.1 *Continued*

postweaning week and by the end of the second week the rate of gain will be at least 1 kg daily in healthy stock. Several days before weaning it is appropriate to remove the mare's daily concentrate allowance and access to hay and pasture may also be restricted.

Epiphysitis (Plate 7.2a–c), probably more correctly called metaphysitis, is not uncommon in faster growing, larger, fine-boned foals of TB, Saddlebred or modern Quarter Horse breeding. It is encountered particularly in the fetlock joint at the end of the metacarpus and in the 'knee' joint at the distal end of the radius. Where it is slight, the foal will probably right matters, but, where severe, supplementary feeds should be restricted to good-quality roughage, the milk intake should be limited and the animal should be boxed until the worst of the 'bumps' subside. A restriction in the rate of weight increase allows joint maturation to continue without the stress of excessive pressure on the joints. Light exercise must then be undertaken daily and the normal feeding regime gradually reinstated. Exercise may, however, be damaging in severe epiphysitis, and in this case no analgesics should be used. Problems of this nature can arise in less than a week and may be complicated by angular deformities in one limb together with epiphysitis in the opposite limb. Successful treatment is contingent on immediate action. Attention by the farrier to the hooves should allow small misalignments of the limbs to be corrected during growth.

Glade *et al.* (1984) have advanced an intriguing explanation of the relationship between epiphysitis and energy and protein consumption at each meal. Excessive intakes suppress normal postprandial hyperthyroxaemia (raised plasma concentra-



(a)

Plate 7.2 Epiphysitis in the lower (distal) end of the metacarpus and the upper (proximal) end of the proximal phalanx (fore fetlock) (a) and (b); and spavin of the hock (c) in foals. (Photograph 7.2 (c) courtesy of Dr Peter Rossdale, FRCVS.)



(b)

Plate 7.2 *Continued*



(c)

Plate 7.2 *Continued*

tion of the thyroid hormone T_4), because an intense insulin secretion stimulates T_3 formation from T_4 , in turn inhibiting TSH (thyrotropin) and thus T_4 secretion. As T_4 is required for bone maturation, hypothyroidism is known to cause skeletal manifestations similar to epiphysitis and OCD, whereas insulin stimulates the formation of immature cartilage. Glade's postulate requires further investigation of the postprandial insulin response, as only 17% hydrolyzable carbohydrate in a diet, given at the rate of 1.6 kg twice daily, was adequate to cause maximum postprandial insulin and glucose responses in TB yearlings (Stanier *et al.* 2002).

Each of these diseases is characterized by enlarged growth centres, failure of bone formation from cartilage, occasional cartilage necrosis and cyst formation. The solution would seem to lie not only in the control of dietary energy and protein and the correction of errors in mineral and trace-element nutrition, but also in raising the number of daily feeds and decreasing their individual size. The logical extension of this may be to change to a system of earlier weaning and feeding foals *ad libitum* a complete mix described under 'Complete mix', Chapter 8. This will encourage nibbling and avoid large postprandial surges in blood glucose and amino acids that stimulate insulin secretion.

Worming of the foal should not coincide with weaning, but the first dose of anthelmintic may be given at two to three months of age or, ideally, at four months of age and thereafter at six-weekly intervals (see Chapter 11).

WEANING PROCEDURE

Restricted feeding of the mare limits milk secretion, but after weaning the udder should not be milked out. Some breeders rub camphorated oil into the udder.

The psychological attachment of the foal to its dam is greatest between the second and 12th weeks of lactation with a peak around the third week, at which time separation leads to the greatest agitation of both. Risk of injury to the foal at weaning as a result of excitement induced by separation is a major factor to be contained. On large stud farms, three alternative procedures are practised:

- (1) All the mares are removed from a year's crop of foals at the same time.
- (2) One or two mares are removed at a time, starting with the first foaled, or most dominant mare, and allowing a few days to elapse before the next is removed.
- (3) The foals are separated for increasing time periods, such that the foals are allowed to nurse three times per day, twice and then only once on successive days, keeping mares and foals in sight of each other.

Methods (1) and (2) may require access to another farm to ensure that weaned stock are out of sight, sound and smell of their mothers. Method (2) may lead to some foals being kicked when searching for milk by more aggressive mares. The last method protracts the drying-up procedure, is more laborious, and not without risk.

It is, however, recommended that, unless there are other mitigating circumstances, mares should be abruptly removed from foals, starting with the mare having the largest and most independent foal, or with the most dominant mare, which is likely to cause problems to other foals. Several days should elapse before the next most dominant mare is removed, leaving the foals in familiar surroundings out of sight, sound and smell of their mothers. It is helpful to leave a gentle dry mare with the foals and any foal having a cold or other debilitating condition should not be weaned until it has regained health.

Foals at first may become frantic and it is important to ensure that all have company, that there is ample space for play, that the pasture has a clean water source and a shelter, and that the shelter and fences are free from protruding nails, splinters and loose wire. The pasture should also be of good quality, without a worm burden, and free from flints and rabbit, or other, holes that might cause leg injury.

Early weaning

The procedures outlined for both liquid and dry feeding of orphaned foals can be followed for early weaning of foals, but the procedure is both labour intensive and an interruption to normal activities on the stud farm. From both a practical and an economic point of view therefore a general recommendation for early weaning may

Table 7.9 Performance of TB foals weaned at 5 days of age (Pagan *et al.* 1993a).

Age (days)	Average BW (kg)	Milk DM intake (kg/day)	Pellet intake (kg/day)	Coarse feed intake (kg/day)	Total DM intake (kg/day)
5–14	72	1.28	0.26	0	1.54
15–28	85	1.34	0.44	0	1.78
29–42	100	1.29	0.63	0	1.92
43–56	115	1.32	0.89	0.10	2.31
57–70	130	1.21	1.00	0.16	2.37
71–84	144	1.25	1.40	0.19	2.84
85–98	158	0.70	1.87	0.51	3.08
99–112	171	0	2.09	0.64	2.73
113–126	182	0	2.03	0.46	2.49
127–140	194	0	2.15	0.47	2.62
141–154	206	0	2.21	0.74	2.95
155–168	220	0	2.27	0.83	3.10

not be given. Nevertheless, there will be circumstances where it has a practical value.

All early-weaned foals must receive colostrum during the first day of life, before they receive other feed. Weaning at three to five days of age, when the mare is not imprinted on the foal, and under hygienic conditions, is safe. A separation of six hours without feed normally eliminates fretting, but initially leads to a lower rate of growth than occurs in foals suckling their dam. The reasons for this are the shock of weaning and a lower milk dry matter intake. In traditional systems, this lower intake is caused by a lower meal frequency than occurs with suckled foals. Compensation by larger meals precipitates diarrhoea. Early-weaned foals should receive a minimum of six meals of milk daily. The milk should contain about 120–130 g DM/l. Eight to ten meals daily would increase the early growth rate without harm; however, retardation of early growth is subject to partial compensation by six months of age. Dry pellets, containing dried skimmed milk and other high-quality protein sources may be offered *ad libitum* during this time. The milk replacer should contain about 25% protein and 16% fat in the dry matter. Some evidence suggests that medium-chain triacylglycerols may be a useful source of fat for foals with a compromised digestive function. Neutral fats, such as coconut oil, that contain medium chain fatty acids are hydrolyzed more rapidly by pancreatic lipase. Any unhydrolyzed medium chain fat is absorbed directly across the small intestinal mucosa. The total daily feed should ideally average 20–25% protein and 12–15% fat. Pagan *et al.* (1993a) reported on the performance of TB foals weaned at five days of age (Table 7.9).

FEEDING THE ORPHAN FOAL

The artificial rearing of relatively small foals should not be lightly attempted. Thoroughbreds of less than 40 kg are normally destroyed. Orphan foals are deprived of

the warmth of the dam and in cold weather should be covered with lightweight quilted material. Normally, a nurse mare is desirable, but in the meantime artificial feeding is necessary. The initial concentration of milk provided should be 22% of dry matter for the first 1–2 days, dropping by 1% daily until a normal concentration of 14–15% of dry matter is reached and maintained until weaning. If there is diarrhoea, the milk can be diluted, or preferably replaced, for a short period with a glucose–electrolyte solution, which provides sodium, potassium, chloride, organic base and glucose in particular (see Table 9.3, p. 328). However, all neonatal foals must receive an adequate source of immunoglobulins following birth before they receive *any other* organic food.

COLOSTRUM

The provision of the newborn with colostral immunoglobulins is crucial to its survival in normal environments. If the mare is lost after the first day, the prospects of foal survival are greatly enhanced as sufficient colostrum should have been sucked by then. Where this is not the case, the maintenance of a bank of frozen colostrum is an asset and its value is enhanced where it has been derived from mares in a similar microbiological environment to that experienced by the foal.

Hygiene in the collection of colostrum is vital, and the preservation process should be carried out by experienced persons to ensure that no bacterial contamination has occurred, otherwise organisms will proliferate when the frozen colostrum is thawed. Thus, minimum quantities, sufficient for each feed, should be stored in each container and then thawed individually. The colostrum should be consumed immediately on warming to preclude undesirable bacterial growth. The foal should receive about 500ml colostrum by nipple or stomach tube every hour for three or four feeds before 12 hours of age. If plasma immunoglobulin concentration can be measured, and it is found to be less than 4g/l, and the foal is less than 12–15 hours old, then 2l colostrum, or an amount to raise plasma levels to 8g/l, should be administered by stomach tube in stages over several hours. Colostrum deprivation in a normal environment inevitably leads to very low serum IgG concentrations and septicaemia with a variety of bacterial species (Robinson *et al.* 1993).

Equine colostrum is the ideal, yet bovine colostrum has a value. Chong *et al.* (1991) raised in isolation 21 of 22 full-term Welsh Mountain pony foals, under conditions free from equid herpesvirus (EVH-1/4), but without mare's colostrum. The foals were given antibiotic prophylaxis and bovine colostrum during the first day. This was followed by mare's milk replacer until weaning. The foals remained free from EHV-1/4 infection.

Evaluation of colostrum

The efficacy of colostrum depends on its IgG content. In the stud, the IgG content can be approximated using a hydrometer, designed for the purpose, that relates the

specific gravity (SG) of the colostrum to its IgG content. Evidence indicates that the estimate is influenced by the fluid temperature, which therefore must be controlled to obtain reliable data. Colostrum with an SG of ≥ 1.085 (equivalent to $>7000\text{mg/dl}$ IgG) at 25°C is suitable for freezing as a source for foals deprived of adequate colostrum from their dams. Colostrum with an IgG concentration of SG >1.03 ($>3000\text{mg/dl}$) at 25°C is considered to be adequate for direct ingestion from the mare's udder within the first 12 hours from birth, so long as no milk has been ingested previously. The IgG level of foal serum should exceed 800mg/dl between 18 hours and 24 hours postfoaling.

Bovine colostrum

Sachets of certified bovine colostrum powder, rich in IgA, IgG and IgM are available commercially and they store well. This powder can be added, preferably to water or the very first feeds of glucose and water, given to newborn foals. These other feeds should *not* be given *before* the colostrum. A reserve supply of this product is a good insurance policy for most studs.

Blood plasma by parenteral or oral dosing

Where the concentration of immunoglobulins in the foal's plasma is less than 400mg/dl and where colostrum is unavailable, one can use blood plasma, preferably from a donor gelding horse or unrelated mare which has never received a blood transfusion and which has been on the farm for some time. The dose is about 20ml/kg BW , given i.v. over a period of one to two hours, that is, an amount totalling approximately 1 l per foal. If the foal trembles, the rate of dosing should be reduced and rapid recovery will soon follow. It should raise the antibody titre of its blood to about 30% of the donor level. Oral dosing with plasma helps in cases of enteritis, but it should normally be given aseptically i.v.

Plasma therapy, given i.v., may be indicated for horses of all ages. It may be necessary in foals exceeding 12–24 hours of age which have received insufficient colostrum and immunoglobulins. It is also frequently indicated for septic foals, or for horses suffering considerable blood loss, where the blood pressure will be low. Therefore a supply of donor plasma held at -20°C is a very convenient source. Frozen plasma is normally thawed in a water bath at 37°C . This is a lengthy process and recent evidence indicates that careful thawing at a defrost setting in a microwave oven, alternating with short periods of agitation, until no ice particles remain, is a satisfactory and quick procedure. The process, carefully handled, causes no apparent damage to plasma proteins.

Taurine

Colostrum contains a high concentration of the non-protein amino acid, taurine. The blood plasma of sick neonatal foals contains low concentrations of this amino

acid, presumably because the undeveloped metabolic system is unable to synthesize taurine from cysteine and cystine, as occurs in the adult. There may be a case for including taurine in foal milk replacer diets, and certainly it is essential to include taurine in colostrum replacers for foals that do not receive colostrum naturally. A dietary concentration of 300 mg/kg dietary DM may be appropriate.

Enteral feeding

The requirement for frequent small feeds has been satisfactorily met by giving low-residue solutions, containing sodium and calcium caseinate, glucose and fat, providing 4.2 kJ/ml through indwelling 12 French enteral feeding tubes. This fluid is given every four hours by gravity flow at the rate of 0.35 ml/kg/min, building up to the total energy needs over four days. However, if gas colic occurs, then an increase in the interval between feeds should help, and drinking water should be offered *ad libitum*. Ousey (1998) stated that foals suffering neonatal maladjustment syndrome achieved, per kg BW, a daily enteral intake of mare's milk, or of milk replacer, only 25% of that achieved by healthy foals of the same age. The unhealthy ones were in negative energy balance, despite their lower activity. Their ME/GE intake ratio was lower than for healthy foals, indicating impaired digestion. Ousey concluded that sick foals should have a minimum GE intake of 260–290 kJ/kg BW/day and that minimal enteral feeding should be combined with parenteral feeding, as deprivation of enteral feeding impairs GI function.

The enteral feeding tubes are placed with the tip in the distal oesophagus and the exterior portion secured by stented suture to the nostril (a stent is a mould for holding a graft in place made of Stent's mass. In this case, it is a device for securing a tubular structure). An extension tube is placed in the free end and secured with a plastic guard to the halter. The tube is flushed with water and capped when not in use. This procedure avoids the risk of trauma to the pharynx caused by frequent intubation. Mineral oil is sometimes added to each feeding, at the rate of 1–4 ml, if constipation occurs, although its use should not persist as it will interrupt the absorption of fat-soluble nutrients.

FEEDING FREQUENCY AND METHOD

Bucket feeding of milk is the traditional method of feeding, although with compromised foals other approaches may be required. The dictum of a little and often applies forcefully to orphan foals. Frequent small amounts reduce the risk of digestive upset and of hypoglycaemia. Where the foal is to be trained to a bucket, the head can be drawn into it with a finger in the mouth – initially this may require the assistance of another person, but soon the foal will adapt to the procedure. All feeding utensils should be clean before each feed. The intake of liquid milk replacer, or a 50:50 mixture of skimmed milk and whole cow's milk, should be at the rate of

280ml every 1.5 hours so that the daily energy intake amounts to 9–10 MJ DE. As a mare's milk replacer, Ousey (1998) considered that goat's milk was tolerated better than cow's milk by neonatal foals, causing fewer digestive disturbances. The GE requirement of healthy neonatal foals was estimated to be 210kJ/kg BW/day, an estimate consistent with that given above. The initial feeding can take place to advantage close by a horse acting as a decoy, but never at the stable door, to avoid the association of it with feed. In order to minimize affinity with man, orphan foals should not be fondled.

Liquid milks are normally given at body temperature, but can equally be given cold. Within a few days the daily intake will attain 9–18l and if the foal is permitted to drink freely it may reach 36l. However, intake should be restricted to a maximum of 18l in a large foal and with any evidence of diarrhoea the quantity should be reduced until the problem has subsided. Once the first few days are over, the liquid can be provided in four then three feeds per day and any excess disposed of.

Automatic liquid-milk feeders of French design have proved very successful on large studs for groups of foals, and they avoid the problem of humanizing. They are electrically operated; the water is warmed and mixed with milk replacer powder at an adjustable rate (Table 7.1). Fresh liquid is prepared to replace that used up as the foals drink. The appropriate concentrations of dry matter suggested above in 'Feeding the orphan foal' should be adhered to, as solutions that are either too weak or too concentrated can precipitate looseness or constipation. New foals are rapidly trained by experienced foals in the same yard.

Creep pellets in the form of stud nuts or concentrate mix, together with milk pellets and a little best-quality leafy hay from seven days of age will encourage dry feeding. Access to *fresh* faeces from a healthy adult horse that has been wormed regularly will provide bacteria of the appropriate kind for seeding the intestinal tract. Any strongyle or ascarid eggs in the faeces should be immature and therefore of low infectivity, and so will passively traverse the GI tract of the foal; but the foal's faeces should be removed regularly. If progress is normal, liquid milk can be discontinued from 30 days of age and intake of dry feed will rise rapidly. At this time the foal may be consuming as much as 2–3 kg dry feed daily, although the consumption of hay will still be rather slight.

FOSTERING

The least troublesome nurse is frequently an old coldblooded mare, especially of piebald breeding, or even a nanny goat. The worst type is a young flighty TB. Prospective individuals should be checked for disease, and their milk should be examined. The udder and the tail should be thoroughly washed and disinfected. The mare can be brought to the stable hooded and disorientated by walking around the area. A strong smelling substance such as camphorated oil can be placed on

the muzzle and the same substance smeared on both the mare's own foal and the orphan. When these two are held together for a while the mare confuses their sounds.

A fostering crate is a boon for nurse mares, enabling foals to be suckled without being kicked. It will have facilities for feeding and watering but the mare should be allowed out for exercise at regular intervals. The crate should have a gate at each end. The critical dimensions are a length of about 250 cm, a width of 65 cm and a gap at both sides of one end 90 × 40 cm, the lower edge of which is 70 cm from the floor for access by the foals to the udder.

THE SICK NEONATAL FOAL

In addition to the tenets of good husbandry there are several important issues upon which action should be taken to further the prospects of the unhealthy neonatal foal.

Immunoglobulin status should be measured. Plasma concentration of IgG should be raised to a minimum of 8 g/l, by administration of approximately 21 colostrum, derived preferably from the dam. This should occur before 12 hours of age. For foals approaching 24 hours of age, or for those that are septicæmic, or hypothermic, 2–4 l of equine plasma, containing at least 1.6 g globulins/l, from a suitable donor, should be administered aseptically and i.v.

Energy reserves are likely to be even lower than those of healthy foals. The risks of hypoglycaemia and hypothermia are considerable. The foal should be held in a warm draught-free box, but with clean air and near its dam.

Following administration of the colostrum (the injection of serum may take place subsequent to a meal of milk/dextrose etc.), milk, preferably derived from the dam, should be given orally (bucket or bottle), or by indwelling nasogastric tube, every one to two hours, so that the 24 hours intake is approximately 20% of the foal's body weight. By remaining within smelling distance of the dam the foal is more likely to be accepted later on, and by milking the dam regularly her supply should not dry up. She is then more likely to suckle her foal at a future date. Fresh drinking water should be available. If the dam's milk is unavailable then a low fat (2%) cow's milk, fortified with 20 g dextrose/l, may be used.

The foal's urine should be monitored for the presence of glucose and the blood should be sampled before several meals to ascertain the status of glucose, TAG, ammonia, urea, potassium and haematocrit. Where TAG is elevated it may indicate hepatic malfunction and fat intake should remain low (with some still present), so long as there is no hyperglycaemia and urinary glucose. With hyperglycaemia the dextrose intake per meal should be reduced to lower urinary glucose level to less than 1%, reducing the risk of renal damage. With elevation in both plasma TAG and glucose a mixture of dextrose and fructose might replace a pure dextrose solution and the frequency of feeding should be increased, giving smaller amounts per meal. Elevated plasma ammonia should be countered with a reduction in milk protein intake. If milk replacer has been used, the milk must have been spray-dried

Table 7.10 Hypertonic i.v. solution for TPN of foals (2.7–3 l/day for a 45 kg foal).

5% amino acid solution	1000 ml
50% dextrose solution	500 ml
KCl	30 mEq
NaHCO ₃	30 mEq
Injectable vitamin preparation	+

* Commercial preparation of fat- and water-soluble vitamins.

and not roller-dried. The detection of an elevated haematocrit and/or elevated plasma albumin concentration can indicate dehydration, compensation for which should be made with Ringer's solution.

If there is evidence of obstruction of the intestines and lack of bowel motility (ileus), impactions or malabsorption, then total parenteral nutrition (TPN) will be necessary. This could be used as an adjunct (parenteral nutrition, PN) to part oral feeding, where digestive ability is poor. For a full account of the problems associated with PN and jugular catheterization the reader is referred to *The Equine Manual* (Higgins & Wright 1995). An i.v. solution for TPN over four days is given in Table 7.10. Alternatively the vitamin supply can be given by sterile intraperitoneal or subcutaneous injection. Monitoring should be as indicated above, including that of electrolyte balance.

Thrombophlebitis is not infrequent and there are several causes. The risk of it may be decreased by complete sterility and absence of septicaemia, the use of the most suitable catheters, the inclusion of heparin in the solution and by replacing approximately half the energy requirement with lipid emulsion (homogenized soya oil, e.g. Intralipid™, Kabi Pharmacia Ltd, Milton Keynes). Lipid clearance should be monitored and fat supplementation should be avoided in cases of liver failure. Lipid use can reduce the risk of hyperglycaemia and it will reduce the total osmolality of the solution. If severe proteinuria is presented, the amino acid (especially glycine) content of the PN solution should be reduced until the renal problem is resolved.

Sugar tolerance

Glucose can be utilized by foals of all ages and maltose is successfully digested and absorbed by foals of four days of age, or more. Intestinal intolerance to specific foods, including lactose, occurs in some foals and older horses. Normal neonatal foals are tolerant of both lactose and glucose, as determined by a rise in blood glucose following oral administration of these sugars (NB: Galactose, a component of lactose, is an epimer of glucose and is rapidly converted to glucose by hepatic UDP (uridine diphosphate)-galactose-4-epimerase). Neonatal foals are, however, intolerant of the disaccharides maltose and sucrose, so these sugars are unsuitable

for inclusion in milk replacers intended for feeding to foals of less than five to seven days of age.

Infections and hygiene

Both respiratory and enteric pathogens are major causes of morbidity in young foals. Browning *et al.* (1991) surveyed 326 diarrhoeic foals in the UK and southern Ireland from 1987 to 1989. They found that Group A rotaviruses were major pathogens in all age groups. Other pathogens included *Aeromonas hydrophilia*, whereas coronavirus, parvovirus, *Campylobacter* spp., *Salmonella* spp. and *Bacteroides fragilis* are currently likely to be minor putative pathogens in Western countries. Rotavirus is a predominant cause of enteritis among foals in the USA. Whether vaccination for this virus may be an effective defence is yet to be established, but disinfection, hygiene and sound management practice will continue to be the primary control measures against the ravages of foal diarrhoea.

Gastric lesions in foals

Gastric lesions among foals showing no signs of gastric disease are of frequent occurrence under ten days of age, but rare over 70 days of age (Murray *et al.* 1990). In common with horses in training, the lesions are situated predominately in the squamous mucosa immediately adjacent to the margo plicatus along the greater curvature. These squamous cells are less extensively protected by mucus than is the glandular mucosa. Lesions were found by Murray and colleagues to occur with lower frequency in the squamous fundus, the glandular fundus and in the lesser curvature. Lesions were more prevalent where there had been diarrhoea, and it has been suggested that a role of GI pathogens, such as rotavirus, may be implicated. However, environmental stress with adrenal medullary hormone secretion, causing mucosal ischaemia, could also be involved.

THE STALLION

The stallion is subject to the same seasonal influences that affect the breeding cycles of the mare: his fertility is greatest in the summer and least in the winter. A large number of studies have been undertaken at Colorado State University to increase our understanding of the mechanisms that control the breeding behaviour of mares and stallions. Reference to their publications should be made where detailed information is sought.

The seasonal changes in the blood concentrations of luteinizing hormone (LH), follicle-stimulating hormone (FSH) and testosterone, and consequential changes in testicular size, sperm production and libido, are functions of changes in photoperiod. Melatonin feed supplementation of stallions July to September in Louisiana caused a reduction in plasma concentrations of the pituitary hormones prolactin and

FSH without influencing testosterone, indicating that melatonin may play an inhibitory role in hypothalamic regulation of the pituitary (Storer *et al.* 2003).

Recrudescence in reproductive activity, as in the mare, is a response to increasing daylight length. Thus, evidence suggests that improved fertility in the early months of the year may be obtained by following a regime similar to that proposed for mares, in which artificial light and richer feed are provided in late December and January. At no time should the stallion be allowed to fatten, and higher fibre, but balanced feeds, are quite satisfactory out of the breeding season. Poorer quality hay supplemented with horse and pony nuts should then be satisfactory, and they will facilitate the imposition of a rising plane of nutrition as the breeding season approaches when stud nuts, or an equivalent concentrate mixture with good-quality hay, should be introduced (Table 7.1). The energy requirements of the stallion rise during the breeding season as a consequence of increased physical activity, in particular that of pacing his run or stall, yet space for physical exercise is important to the stallion in all seasons.

There is little evidence to support the use of special supplements to enhance the fertility of stallions, but a diet of 0.75–1.5 kg cereal-based concentrates plus hay per 100 kg BW daily and clean water (Table 7.5) should suffice.

Artificial insemination

This subject is outside the province of this text, but some nutritional information is available. Bruemmer *et al.* (2002) concluded that if stallion spermatozoa are cooled and stored for longer than 24 hours their fertilizing capacity declines. However, if 2 mM pyruvate and skim milk are added to cooled spermatozoa, oxidative stress is reduced and motility and fertility are maintained for 48 hours.

STUDY QUESTIONS

- (1) What issues should be considered in deciding the optimum weaning age for a stud and for an individual foal?
- (2) On making arrangements and provisions for colostrum-deprived foals what are the important issues?
- (3) What action would you take in the event of diarrhoea occurring:
 - (a) in a suckled foal;
 - (b) in a weaned foal; and
 - (c) when there is an outbreak among several foals of similar age?

FURTHER READING

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Chapter 8

Growth

For it is easy to demonstrate that a horse may irrecoverably suffer in his shape and outward beauty as well as in strength by being underfed while he is young.

W. Gibson 1726

Normal growth patterns, growth quality and conformation in the horse are to a considerable extent beyond the control of the stud. Nevertheless, breeding and diet play increasingly measurable roles; consequently, their contribution will undoubtedly accelerate. A key to that input is an agreement of objectives that can be clearly characterized and measured.

IDEAL CONFORMATION

Selection of breeding partners for shape and size has undoubtedly concerned breeders since domestic horse husbandry began. However, the shape of a horse that is required to maximize its performance in the type of activity for which it has been bred has received scant objective attention. Variability in shape within breed is considerable. Mawdsley (1993) found a wide variation in linear traits within TBs, providing possible scope for progress by performance testing. (Variation in tissue type for work is discussed in Chapter 9.) In order that progress is achieved to improve the performance potential and working lifespan of foals it is necessary:

- for measurable objectives to be agreed within breeds at specified ages;
- for progeny performance testing to be conducted; and
- that allowance is made for corrections to malformations by the farrier and others.

How does normal growth unfold?

BIRTH WEIGHT AND EARLY GROWTH

Growth proceeds through a process of cell division and enlargement initiated by the fertilization of the egg. Cells differentiate, forming the various embryonic tissues. Soon after birth the number of cells in most tissues has reached a maximum and

further growth is accomplished by hypertrophy (enlargement) of the individual cells; but in some tissues, for instance epithelial tissue, cell replication continues throughout life in order to replace cells that are sloughed off, or, in hepatic tissue, to compensate for malfunction, for instance where neoplastic disease is present. Not all tissues, organs and structures increase in size at the same rate, so that during growth the shape of the animal changes.

The potential for a maximum rate of growth, measured in kilograms of daily body weight gain, persists until about nine months of age in the horse, at which time it gradually declines and ceases as the adult size and shape are attained. However, the overall rate of growth measured relative to the existing weight, i.e. say at 50 days of age $(\text{kg daily gain})_{150}/(\text{BW}_{150})$, is initially slow (Fig. 8.1); it accelerates to a maximum before birth and then declines. By the seventh month of gestation, merely 17% of the birth weight and only 10–15% of the birth dry matter have been accumulated. Thus, the accretion of most of the energy and minerals present at birth occurs during the last months of gestation.

Partly because the mature number of cells in many tissues of the adult has been achieved by birth or shortly afterwards, the maximum adult weight of horses and ponies is, to a large extent, determined by birth weight. As a rough guide, birth weight constitutes 10% of the adult weight. Among TBs, individuals weighing less than about 35 kg at birth are very unlikely to reach 152 cm (15 hands) in adult life. One study revealed that the proportion of foals with a birth weight of less than 40 kg that actually raced was much smaller than the proportion of those weighing more than 40 kg (Platt 1978). Horses out of small mares by small stallions will be small as adults, but will achieve their mature size slightly sooner than will the products of large parents. Differences between breeds in rate of attainment of mature weight are greater than the differences in rate of attainment of mature height.

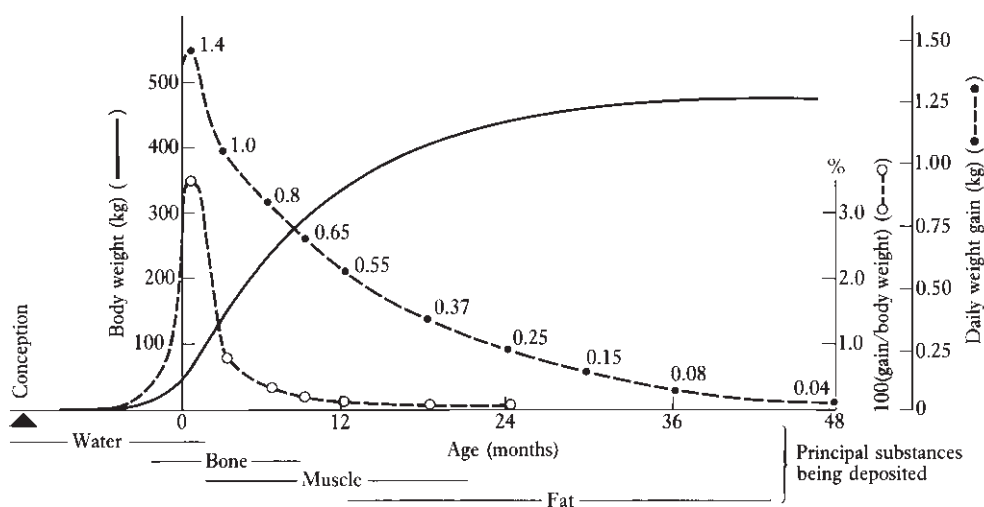


Fig. 8.1 Normal growth curve for the horse (mature weight of example, 500 kg).

Table 8.1 Percentage of mature body weight and of withers height attained at different ages in various horse breeds (Hintz 1980a).

Age	6 months		12 months		18 months	
	Weight	Height	Weight	Height	Weight	Height
Shetland pony	52	86	73	94	83	97
Quarter Horse	44	84	66	91	80	95
Anglo-Arab	45	83	67	92	81	95
Arabian	46	84	66	91	80	95
TB	46	84	66	90	80	95
Percheron	40	79	59	89	74	92

Studies being undertaken at the time of writing at the Thoroughbred Breeders' Association Equine Fertility Unit in Newmarket, at the Royal Stables in the United Arab Emirates (UAE) and at The Sheikh Mohammed Camel Reproduction Laboratory, Dubai, should eventually throw much more light on the effect of foetal size on adult size, extending the original research of John Hammond, at Cambridge in the 1940s and 50s. The work in the UAE includes crosses in both directions between llama and camel, embryo transfer and implantation in foster camels and implantation of Arabian embryos in draught mares.

Table 8.1 gives data for several breeds of horses. As a general rule, foals attain 60% of their mature weight, 90% of their mature height and 95% of their eventual bone growth by 12 months of age. In the period from birth to maturity, the growth coefficients (the rate constant for the growth of a tissue, or structure, relative to that for the whole empty body) of the three major equine tissues in order of increasing magnitude are bone, muscle and fat, implying that bone is the earliest maturing tissue and fat the latest. This, and the earlier assertion that overall growth rate declines from birth (weight gain per unit empty live weight), signifies that ultimate height is determined in very early life, that early growth demands diets rich in bone-forming minerals, protein and vitamins, and that with increasing age an increasing proportion of dietary carbohydrate is required. The early rapid extension of the long bones of the legs, pronounced in tall breeds, render them subject to deformation through early malnutrition.

Early nutrition and weaning age

The nutrition of the foal at birth is affected not only by the feeding of the mare but also by the physiological efficiency of the uterine environment. This may to some extent be affected by the age of the mare, as indicated by the data in Table 8.2. Differences in birth weight brought about by nutritional deviations in the pregnant mare can, however, be proportionately lessened by nutritional adjustments in early postnatal life. Although birth weight has a major impact on ultimate size for both genetic and environmental reasons, weaning age, in conditions of good manage-

Table 8.2 Effect of age of TB mare on body weight and height at withers of foals (Hintz 1980a; data based on 1992 foals).

Age of mare (years)	Age of foal			
	30 days		540 days	
	Foal weight (kg)	Height (cm)	Weight (kg)	Height (cm)
3–7	93.0	108.0	393.7	152.4
8–12	97.5	110.5	401.4	153.7
13–16	98.0	110.5	396.9	153.0
17–20	95.3	109.2	391.0	152.4

Table 8.3 Effect of sex on growth of TB foals (Hintz 1980a; data based on 1992 foals).

Age (days)	Body weight (kg)		Withers height (cm)	
	Colts	Fillies	Colts	Fillies
2	52.2	51.3	100.3	99.7
60	136.5	134.7	118.3	118.1
180	244.9	235.9	134.6	133.0
540	435.5	401.4	154.3	152.4

ment, has little influence. Foals weaned after receiving colostrum can achieve growth rates equal to that of those weaned at two to four months of age. These in turn may grow faster than foals weaned at six months of age. The appropriate weaning age, therefore, for any particular stud farm, turns on the most convenient and reliable management practice.

LATER GROWTH AND CONFORMATIONAL CHANGES

Initial and ultimate weights and heights differ as between colts and fillies (Table 8.3). The differences are small and limited studies in England (Green 1969) failed to detect sex differences in linear measurements, or any between early- and late-born foals. Nonetheless, some substantial evidence suggests that foals born late in the season are heavier and taller than early-born foals (Table 8.4) despite a somewhat shorter period of gestation. As previously indicated (Chapter 7), gestation length seems to be a function of daylight length in late gestation. Birth weight and subsequent rates of growth, as implied above in this chapter, also depend on the mature size of the breed. Micol & Martin-Rosset (1995) recorded that the rate of weight gain from birth to weaning on pasture of the heavy French breeds – Breton, Comtois and Ardennais (700–800 kg mature BW) is 1.3–1.7 kg/day. Even under harsh upland conditions the foals gain at the rate of 1.3–1.5 kg/day.

Table 8.4 Effect of month of birth on body weight and withers height of TBs (Hintz 1980a; data based on 1992 foals).

Month of birth	Age			
	30 days		540 days	
	Weight (kg)	Height (cm)	Weight (kg)	Height (cm)
February–March	95.3	109.2	396.9	153.7
April	97.5	110.5	402.8	153.7
May	100.7	111.1	403.7	153.7

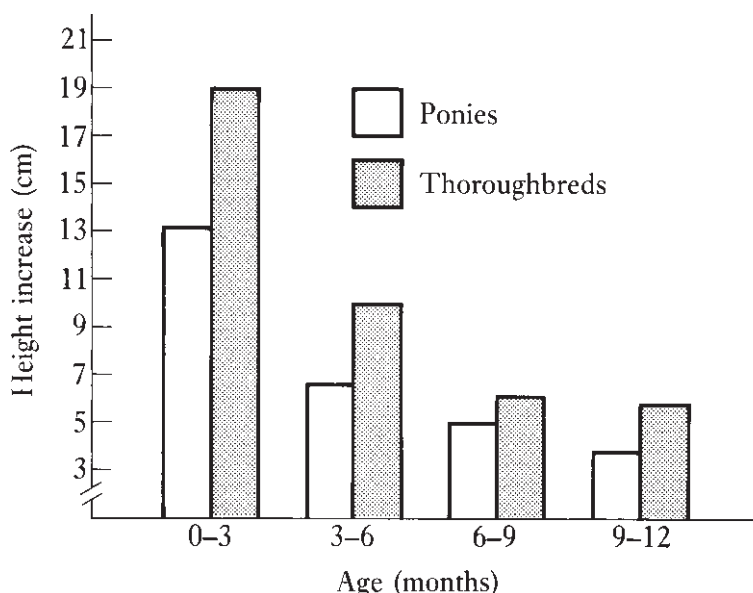


Fig. 8.2 Height increase (cm) at withers of pony and TB foals (Campbell & Lee 1981).

On the whole, differences in growth rate, after the neonatal and postweaning periods, have little influence on subsequent growth, or skeletal measurements, in TB and Quarter Horse foals (Peterson *et al.* 2003) or on mature size. Lawrence *et al.* (2003a) also estimated that the variation in growth rate within the Morgan horse population had little impact on their ultimate height. Although maximum height may be approached soon after 12 months of age, this may be delayed without a reduction in the ultimate measurement by reducing the rate of feeding slightly. Similarly, 90% of mature weight may be achieved at 18 months, but delayed until 24 months by the same restriction. In Fig. 8.2 the changes in height at the withers over the first 12 months of pony and TB foals are compared. Although the foals achieve very different heights, the pattern of growth is similar.

Height at the withers largely reflects linear growth of the long bones in the front legs. Long bones increase in diameter or thickness throughout their length, but no

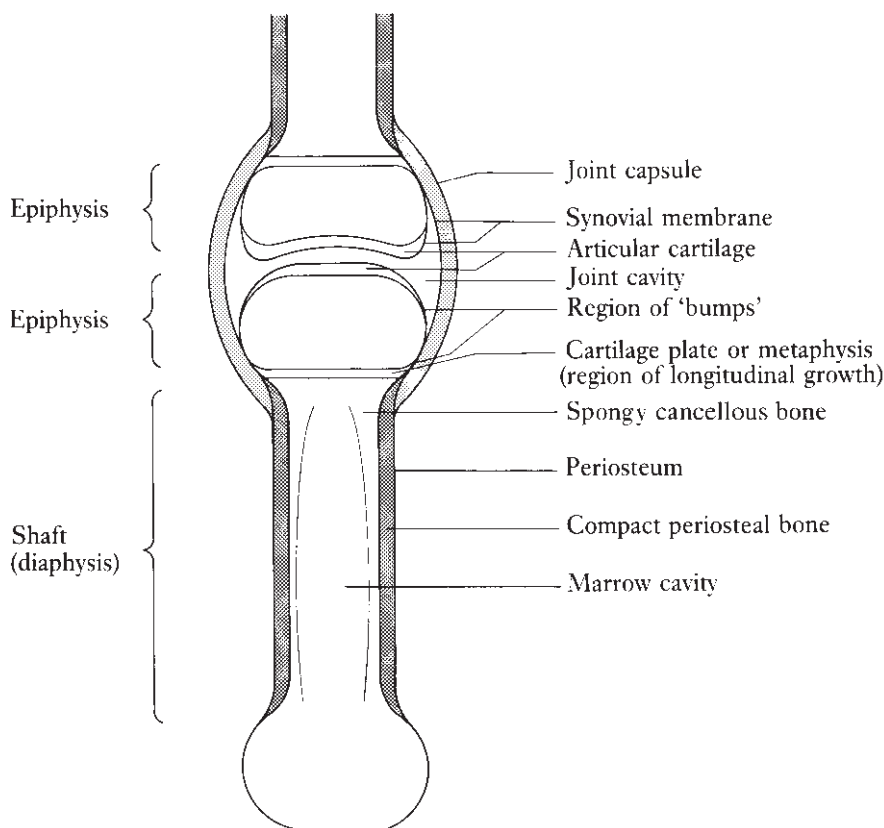


Fig. 8.3 Long bones and their articulation in late growth (joint cavity has been expanded for purposes of visualization). Note the regions of growth: cartilage at either end of diaphysis; cartilage of epiphysis; and periosteum of shank.

increase in length occurs within the shank, or diaphysis, after birth (Figs 8.3 and 8.4). They increase in length by growth in a metaphyseal plate at both the near and distant ends (proximal and distal) from the body. The rate of growth at each end is different. Table 8.5 gives values recorded for crossbred ponies (Campbell & Lee 1981). Correction of distortions in bone growth, owing to bad feeding practices and other causes, is possible during the phase of rapid growth of the end in question. Thus, for the distal radius or tibia, such correction could be imposed up to 60 weeks of age, whereas fetlock distortion, a fairly common condition, requires treatment by three months. In either case, temporarily restricted growth will either not or only slightly influence ultimate size (see 'Developmental orthopaedic disease'; also see below, Brauer *et al.* 1999).

In recent years it has been determined that the bone-specific protein, osteocalcin, is an indicator of osteoid production and its serum concentration is increased during growth and bone matrix formation. This concentration increases with increased bone turnover (mobilization and formation). (Other biochemical markers of bone

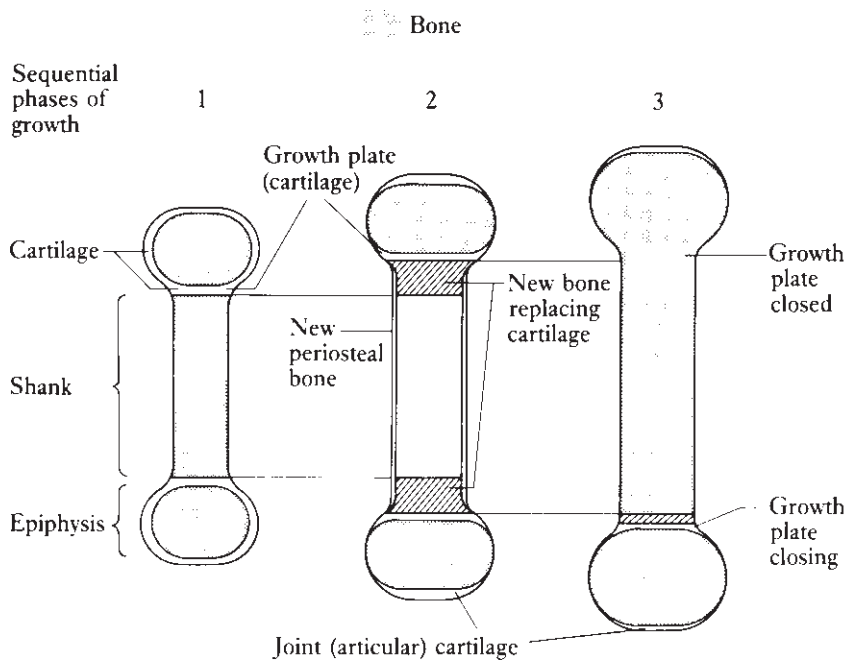


Fig. 8.4 Typical growth of long bone, for example radius or tibia (after Rossdale & Ricketts 1980).

Table 8.5 Growth of leg bones in three male and three female crossbred ponies (Campbell & Lee 1981).

Bone	Length of bone (cm)		Increase at each end (%)		Age at closure of growth plates (weeks)	
	0-7 days	2 years	Proximal	Distal	Proximal	Distal
Femur	21.6	30.9	24	20	55	55
Tibia	21.3	29.1	22	17	60	60
Radius	20.7	28.1	12	24	54	69
Humerus	16.0	22.6	31	11	80	70
Metatarsal	22.7	23.7	5	5	40-44	40-44
Metacarpal	18.4	19.4	5	5	40-44	40-44
Phalanges 1	5.9	6.6				
Phalanges 2	2.3	2.5				
Phalanges 3	5.6	6.2				

formation include type I collagen carboxy-terminal propeptide, and the bone-specific isoenzyme of alkaline phosphatase. Cross-linked telopeptide of type I collagen is a marker of bone resorption and N-terminal propeptide of type III collagen is a marker of soft tissue turnover, Price *et al.* 2001). Although a low dietary cation-anion balance (DCAD) is expected to increase bone mobilization (resorption) and urinary loss of Ca, an adverse effect on skeletal growth may not occur (see below 'Minerals, trace elements and DOD'; Cooper *et al.* 1999a).

The balanced adult horse of good conformation has a height at the withers that equals its length from the tip of the shoulder to the point of the buttocks (see Fig. 6.1, p. 187). By contrast, a foal is taller than it is long (Plate 8.1) so that it inevitably trots wide behind. Fillies at birth tend to be fairly level across the top, but may be as much as 5 cm higher over the croup than over the withers at one year and then balanced again by five years. The length of their bodies tends to be greater than their height at the withers. Colts are generally higher over the hip at birth but level by three years. Some horses that have grown poorly in the front legs are lower over the withers than the croup at maturity and, as nearly 60% of the body weight is normally carried by the front legs, this unusual conformation can force additional weight and stress on the forequarter, increasing the risk of damage.

Similar height increases at the withers and at the hip are found in TB foals between 14 and 588 days of age when given feed at recommended rates. The hip height is about 1 cm greater than the withers height throughout this period (Thompson 1995). Cymbaluk and colleagues (1990) found that Quarter Horses fed *ad libitum* cf. limit-fed at 6–24 months may have a slight tendency to gain relatively more in hind-body mass than in fore-body mass, but to gain slightly less in croup height than in withers height. Thus, adult conformation could be influenced by rate of feeding during growth, and very restricted feeding may restrict development of the hindquarter powerhouse. The extent to which compensatory growth occurs after 24 months is unclear.

When fed liberally, the growth of ponies up to nine months of age may increase to nearly 1.5 kg/day, although by 12 months the rate may have fallen to half this amount. Under hill conditions, where young stock can receive little in the way of concentrates, growth can be strikingly depressed. Morgan horses on range were shown to be between 23 and 46 kg lighter and 2.5 cm shorter at five years of age than their contemporaries in the same environment receiving supplementary cereal-based concentrates (Dawson *et al.* 1945). With only moderate restrictions, such horses may attain normal mature height at five years, whereas with more liberal feeding they can reach it by three years (Kownacki 1983). New Forest filly ponies, fed from six months of age throughout the winter at a rate that only maintained body weight, grew faster during the following summer on rough pasture than their counterparts permitted to gain 0.4 kg/day in the winter (Ellis & Lawrence 1978a,b, 1979, 1980). Although body weight may remain constant in such deprived circumstances, parts of the skeleton grow differentially so that the restricted fillies in this example were tall, thin and shallow-bodied. With a delay in the closure of growth plates these differences are partly corrected during later growth. Catch-up growth of this kind is not without risk among potentially tall breeds, and although the evidence against interrupted growth curves is slight, a smooth growth curve should be sought for TBs, Saddlebreds and the like (Fig. 8.1).

It is concluded that compensatory growth occurs in horses with some small influence on ultimate conformation, but the influence of this on the extent of active working life is yet far from clear. The subject of growth abnormalities is discussed in 'Developmental orthopaedic disease', this chapter.



Plate 8.1 Grey colt (51 kg at birth) by Vitiges out of Castle Moon at Derisley Wood Stud, Newmarket: 5 days old (top) and 82 days old (below). Note that the height of the young foal exceeds its length, a characteristic that changes as adult proportions are attained. Over a period of as little as 77 days the length of the foal has increased considerably so the body proportions are already more like those of the adult. The height of the croup tends to increase faster than that of the withers during the first few months, but by three years of age in the TB the withers height has caught up, through differential growth of the leg bones.

Supplementary feeding in the first winter

With full access to good summer pastures, yearlings require no supplementary feed (that is, assuming the land has no specific trace-element deficiencies). The amounts of supplementary feed necessary during the previous winter will depend, first, on the quality and, second, on the amount of grazing available. Winter pastures have little value as a stimulus to growth in young stock, but will provide reasonable roughage to complement cereal-based concentrates. Until the spring flush is available, stock at this age should receive from 1.25 to 1.5 kg concentrates containing 16% protein and appropriate vitamins and minerals per 100 kg BW daily (see Figs 6.2–6.4, pp. 188–190, for height and girth equivalents).

When exercise is limited by in-wintering, nuts or other concentrate feeds should be given to young stock in two meals daily to avoid intestinal upsets that might cause oedema in the legs. Stabled, weaned horses may be given free access to good-quality hay, and when they are receiving concentrates at the rate of 1.25–1.5% of body weight, they will consume hay or other forage dry matter at a rate of about 0.5–1.5% of body weight. Using good-quality hay, Ott & Kivipelto (2003) found similar growth rates (0.75 kg/day) and bone formation amongst TB and Quarter Horse weanlings when concentrate:hay ratios were 1:1 or 1.7:1.0 in the period from weaning at 112 to 224 days of age. In a study of New Forest and Welsh ponies, the maintenance requirement of the filly foals amounted to 14–14.6 MJ DE/100 kg BW, equivalent to 1.75 kg average hay or 1.1–1.2 kg average nuts per 100 kg BW (Ellis & Lawrence 1980). At the end of the first winter, that is, when yearlings are 11 or 12 months old, colts and fillies should be separated. The first oestrus should occur before May, unless winter conditions and feed have been poor.

Long yearlings

From 90% of mature weight long yearlings (during their second year of life when 80–90% of mature weight has been attained by light breeds of horses) can be fed most economically on maintenance rations as for mature horses. This should include good-quality hay and a concentrate offered at a rate of 0.75–1% of body weight and providing cereals, a concentrate protein source, minerals and vitamins. However, when these more slowly growing stock are out on good-quality pasture continuously they should need no other source of feed, apart from a trace-element mixture should a specific deficiency exist.

Meat production

The heavy breeds [Breton, Comtois and Ardennais (700–800 kg mature BW)] are grown in France for meat in high forage systems. The systems include: (a) colts castrated at 18 months and surplus fillies slaughtered at weights exceeding 700 kg off grassland in mixed systems with cattle; (b) following grazing and finishing indoors at 10–15 months of age (450–500 kg) BW with continuous gains of 1.0–1.4 kg/day; (c)

slaughtered supplemented with cereals on grassland at the end of the grazing season at 18 months (550–580 kg BW); or (d) at 22–24 months (620–670 kg BW) after finishing indoors during the second winter. The interesting observation was made by Micol & Martin-Rosset (1995) that as concentrate allowance to finishing horses is increased, the *ad libitum* intake of hay decreases by 1.26 kg DM for each 1.0 kg increase in concentrate. On the other hand, where maize silage is given (>30% DM), the silage DM intake decreases by 0.81 kg for each 1.0 kg increase in concentrate, so overall intake increases. The concentrate improves silage acceptability as the horses consume 24 g DM/kg BW daily of a 100% hay diet, but only 16 g DM/kg BW daily of the 100% silage diet. The concentrate contained (g/kg): 900 ground barley and maize, 80 soya-bean meal and 20 minerals and vitamins. It was given in some studies 50:50 with maize silage or hay (Martin-Rosset & Dulphy 1987).

EFFECTS OF DIETARY COMPOSITION

Nutrient requirements are dealt with in Chapter 6. Certain aspects of those requirements that have engaged the interests of research workers over the last fifteen years are considered here, especially where it may affect the quality of the skeleton and mobility of the horse.

Dietary protein

The NRC (1989) has published dietary crude protein requirements for growing horses. The needs of the horse are for protein that is digested, yielding products that are absorbed. The digestibility of proteins differs to a small extent between sources. Digestibility is normally expressed as apparent digestibility, which is:

$$\frac{(\text{N intake} - \text{faecal N})}{\text{N intake}}$$

As endogenous faecal N loss is proportional to body weight and not to N intake

$$52 \text{ mg N} \times \text{kg BW}^{0.75}$$

(Slade *et al.* 1970), apparent N digestibility increases as the N content of the diet is increased. By comparison, true digestibility, for which the endogenous N loss is subtracted from the faecal N, is similar for a range of dietary N concentrations. For most protein sources, true digestibility falls in the range 0.7–0.9 and for most common sources it is in the range 0.75–0.85.

As discussed previously, true digestibility of protein should refer to that occurring in the small intestine only, to exclude non-protein N absorbed from the large gut. Within practical limits, dietary concentration of protein has no material influence on the proportion of amino acids absorbed from the small intestine. Amino acid digestibility should be central to formulation methods, as concentrate proteins generally

are more valuable in this respect than are forage proteins of the same amino acid composition.

Three diets, containing all nutrients at concentrations recommended by the NRC (1978), except protein, have been compared in Arabian, TB and Standardbred foals from four months of age (Schryver *et al.* 1987). The dietary protein and lysine concentrations were: 90, 140 and 200 g/kg and 2.8, 7 and 12.6 g/kg diet DM, respectively. The lowest protein intake depressed the rate of daily weight and height gain and gain in forecannon circumference, whereas there was no significant difference in the responses to the other two diets. At nine months of age, the foals that had received the 90 g protein/kg diet were changed to 200 g. After a further 140 days there was no significant difference in body weight, height or cannon circumference among the groups. The NRC (1989) recommends 131 g protein/kg diet (90% DM, i.e. 146 g/kg DM) from four to six months of age. Breeders have frequently limited the protein content of the weaned foal's diet to avoid the development of skeletal defects. Nevertheless, there is little evidence that high intakes of protein are a cause of this frequent problem in several breeds (see 'Developmental orthopaedic disease', this chapter).

In the experiment of Schryver and colleagues, rate of feed intake, body weight gain, height gain and the increase in forecannon bone girth were all greatest with the highest protein intake, although differing not significantly from the 140 g diet. Higher protein and lysine allowances increase cannon girth, whereas inadequate protein intake tends to produce more spindly bones that are only slightly shorter. Some evidence, however, does indicate that bone density is slightly lower in foals that are allowed to grow at their maximum rate, and therefore bone strength for a given body size may be slightly compromised by very rapid growth rates.

With growing horses of all ages it is important that the energy allowance is not excessive with respect to the protein allowance. Commencing at 120 days of age, poorer body condition has been observed in foals given 125% of the recommended NRC (1978) energy allowance but only 100% of the protein allowance, compared with foals given 100% or 125% of both NRC energy and protein allowances. Possibly as a result of this and other evidence the NRC (1989) recommendation for the protein allowance of yearlings is 20% greater than the earlier recommendation, although for weanlings a similar recommendation is given. Most evidence also indicates that increased growth rates can be achieved by providing more dietary protein, of average quality, than the amount recommended by the NRC (1978). On the other hand, the INRA (French) recommendation, given as MADC (see Chapter 6), is 66% of the NRC (1989) crude protein value at 6 months and only 54% at 12 months. Very approximately the MADC value should be 70% of the crude protein value to be equivalent. The crucial factor is which proposal leads to the healthier ultimate development of the frame.

Grace *et al.* (1998a) observed that filly yearlings with an initial weight of 300 kg, gained at an average of 0.75 kg/day on a New Zealand pasture containing 201 g crude protein/kg DM. Their average DM intake was 6.5–7.5 kg/day. The pasture contained 11.4 MJ DE/kg DM, so the daily DE intake was 81 MJ DE, which was considered by

the authors to be adequate. There was, indeed, abundant high-quality protein available in relation to digestible energy, yet amino acid digestibility was an unknown.

In experiments by Thompson *et al.* (1988) energy, protein, Ca and P intakes, relative to NRC (1978) recommendations, were varied in the diet of growing foals. The results showed that inadequate dietary Ca caused lower increases in bone-mineral content and in long-bone length. Raised intakes of energy, or of protein, increased the rate of growth in length of several bones; but inadequate intakes of protein relative to that of energy compromised the development of cortical bone and led to smaller increases in withers height. Thus, fast rates of body-weight increase caused by excessive energy intakes introduce an increased risk of inadequate bone development, where the balance of nutrients is incorrect, increasing the risk of conformational and musculoskeletal abnormalities (Thompson *et al.* 1988; Cymbaluk *et al.* 1990).

In the experiment of Schryver *et al.* (1987) Ca metabolism was not adversely affected by high protein intakes, even though the 200 g protein diet contained nearly twice as much sulphur (S) as was contained in the 90 g protein diet. Other evidence indicates that a protein intake that is 130% of the NRC (1978) recommendation for foals causes an increase in the urinary excretion of Ca and P. This is primarily the result of the increased S-amino acid consumption causing a decrease in renal tubular filtrate pH and a consequential reduction in the renal tubular reabsorption of Ca and P (see cation-anion balance, Chapters 9 and 11 and Appendix D). The difference between studies in the risk of urinary Ca loss may be due to:

- (1) differences between studies in the actual acid:base balance of the diets; and
- (2) the age at measurement. There is a more rapid turnover of the amorphous calcium phosphate salts, found in greater abundance in the bones of younger animals, compared with the turnover of the more stable apatitic crystals that predominate in the bones of older animals.

There is therefore a theoretical risk to bone calcification and dietary supplementation with sodium bicarbonate may be indicated with high protein diets. Optimum amino acid balance of these diets is also indicated, as excessive imbalanced protein could exacerbate urinary Ca loss.

Limiting amino acids

The first and second dietary limiting amino acids for the growth of foals are lysine and threonine, when they are given a diet of maize and oats, in equal amounts, plus soya, dried lucerne and coastal Bermuda grass hay (Graham *et al.* 1994). Staniar *et al.* (2001) demonstrated that a supplement of lysine and threonine, included at the rates of 6 g/kg and 4 g/kg respectively in a 9% protein concentrate and given to growing foals, surprisingly increased their weight gain on winter pasture, compared with the response to a concentrate containing 14% protein and 22% soya-bean meal.

An excessive consumption of these amino acids does not materially compromise the tubular reabsorption of Ca. The lysine requirement, as a proportion of the diet, decreases rapidly with increasing age, but in the Finnhorse breed it exceeds 31 g/day up to ten months and it is important for normal blood haemoglobin and red blood cell formation (Saastamoinen & Koskinen 1993). An imbalance in dietary amino acids is likely to increase urinary losses of S, for a given rate of N retention, and therefore, as indicated in 'Dietary protein' above, excessive poor-quality dietary protein may compromise Ca balance.

Summer pasture generally contains more protein, and of a higher quality, than protein given to in-wintered horses. If inadequate dietary protein is given to foals during their first winter, Scandinavian evidence indicates that there is a fall in the concentrations of plasma albumin (Mäenpää *et al.* 1988a,b). According to this evidence, total plasma protein fell from 68 g/l in November to 56 g/l in April and reductions occurred in the plasma concentration of several of the free essential amino acids: isoleucine, leucine, lysine, phenylalanine, threonine and valine. Perhaps more consistently under these conditions, decreases in plasma lysine, methionine and valine have been reported (Saastamoinen & Koskinen 1993). These decreases can be associated with a marked interruption of the normal pattern of growth to 12 months of age. On the other hand, other reports indicate that the effect of inadequate dietary protein on serum total protein has been slight, or absent, in adult ponies (Reitnour & Salsbury 1976) and among weanlings, when growth rate has been influenced (Godbee & Slade 1981), although Saastamoinen & Koskinen (1993) also found a reduction in the concentration of blood haemoglobin of their in-wintered foals.

Complete feed mixtures for growing horses

Complete mixtures of concentrates and chopped roughage are routinely given *ad libitum* to growing cattle, but to suggest that similar mixtures should be given to groups of growing TBs and horses of other breeds may seem an affront, or at the very least inappropriate. When a number of such stock are reared together through both the first and even the second winter months, there is much to recommend such a procedure. It does require sheltered *ad libitum* feed hoppers, as by ensuring that the feed is always present, risk of colic or of laminitis (founder) is nonexistent in foals. Complete feed mixtures should initially contain a cereal-based concentrate and good-quality chopped hay in a ratio of 2:1, gradually falling, as the growth rate declines, to a ratio of 1:1. Stock will normally consume a total amount of dry feed daily equivalent to 3–3.5% of their body weight. The physical make-up of the mix and particle size should be so regulated that no segregation between the hay and the concentrate arises. Molasses is often a good material to include in the mix as an aid in preventing segregation and some ingenuity is required to ensure that no bridging occurs in hoppers. The author's own evidence (Frape 1989) with such mixtures has been very encouraging, as assessed by conformation and control of developmental orthopaedic disease (DOD).

Preparation for sale

TB yearlings are normally prepared for the autumn sales by excluding them from pasture. A diet based on 1.5 kg good-quality stud nuts or other concentrate, plus 1–1.5 kg hay/100 kg BW is a typical practice. Traditional concentrate mixtures contain 75–90% of oats, plus bran, soya-bean meal, dried skimmed milk and sometimes a vitamin/mineral premix. There is no particular reason, however, why these horses should not be provided with a complete mixture of concentrates and chopped hay as advocated for younger stock, but in a ratio of 1:1 concentrates to hay. Table 8.6 gives proposed feed mixtures for growing horses and Table 8.7 rates of feeding (on the basis of 90% DM). The composition of these diets conforms with the principles of equine growth previously propounded in this chapter.

Table 8.6 Feed mixture for growing horses for providing with chopped forage or grass hay or as a supplement to poor pasture.

	Concentrate mixtures (%)		
	Weaned foal	Yearling	Presale of long yearling
Oats	41.1	44.7	59.0
Wheat bran	15.0	15.0	10.0
High protein grass meal	15.0	15.0	10.0
Extracted soya-bean meal	18.0	15.0	10.0
Molasses	7.5	7.5	7.5
Feed-grade fat	1.0	1.0	2.0
Limestone	1.2	0.7	0.5
Dicalcium phosphate	0.5	0.5	0.4
Salt	0.5	0.5	0.5
Vitamins/trace elements*	0.2	0.1	0.1
Total	100.0	100.0	100.0

* See Table 7.1, p. 249.

Table 8.7 Daily feed allowances (kg) for growing horses (500 kg mature weight).

	Concentrates	Hay (clover/grass)
<i>Foals</i> (kg BW)		
100 (4–5 weeks)	0.5	—
130–180	2.2–3.2	1.3–1.9
180–230	2.9–3.9	1.8–2.3
230–270	3.6–4.8	2.2–2.8
270–320	4.0–4.6	2.7–3.2
<i>Yearlings</i> (kg BW)		
310–360	3.5–4.5	3.0–3.7
360–410	3.0–4.2	3.6–4.1
410–460	3.0–3.8	4.0–4.5

DEVELOPMENTAL ORTHOPAEDIC DISEASE (DOD)

Disturbances of skeletal growth and development in the horse have been included within the umbrella term developmental orthopaedic disease (DOD) (Jeffcott 1991). The major disorders included are:

- dyschondroplasia (DCP) (or osteochondrosis [OC]). Osteochondritis dissecans (OCD) is a similar condition, but occurs where inflammation is present and a separation of a piece of articular cartilage and of the underlying bone within the joint has occurred. Thus, subchondral bone plays a role in the pathogenesis of osteochondral damage and osteoarthritis (Kawcak *et al.* 2001);
- physitis (physeal dysplasia or epiphysitis);
- angular limb deformities and flexural deformity (contracted tendons); and
- vertebral abnormalities (wobbler disease; see also 'Vitamin E', Chapter 4).

The likely causes of DOD include:

- congenital malpositioning and/or malnutrition;
- high rates of postnatal growth with unbalanced diets;
- probably biomechanical trauma;
- endocrinological dysfunction;
- toxicity, including iodine toxicosis; and
- heredity. Many investigators have demonstrated this in several breeds (Sandgren 1993). NB: whereas most colts with signs are destroyed, most fillies are retained. Sire index is related to DOD, yet stallions having a propensity to sire affected offspring may not show signs themselves.

DOD has a wide distribution and frequency in horses. Radiographic signs of joint disease in the distal tarsal joints were found by Björnsdóttir *et al.* (2000) in 30.3% of Icelandic riding horses. Dyschondroplasia (DCP) is a generalized disease of synovial joints in young animals resulting from a disturbance of the endochondral ossification of the growth cartilage, involving secondary osseous remodelling. It therefore embraces lesions in the articular, epiphyseal and metaphyseal growth-plate regions of long bones. When it affects the articular epiphyseal cartilage, it may result in synovitis.

The condition is believed to begin as an abnormality of chondrocyte development and maturation and to extend to an alteration in their matrix secretion, culminating in altered endochondral ossification (DCP). Reactive oxygen species are capable of degrading many components of the joint in the presence of insufficient antioxidant defences. Dimock *et al.* (2000) found a higher amino-acid residue carbonyl (ketone) content and a marginally higher antioxidant content of the diseased synovial fluid protein in leg joints. Whether a higher carbonyl level is a precursor to damage has to be determined, as oxidative damage increases during the disease process and may accelerate it. The higher antioxidant status of the diseased joints may only reflect an increase in the acute-phase protein content during an inflammatory response.

The proliferating chondrocytes produce and secrete the extracellular cartilage matrix of proteoglycans and collagen type II. In contrast to the proteoglycan component, the collagen component of cartilage is known to have very limited capacity for repair, owing to an extremely low turnover rate. Brama *et al.* (2000), using two-year-old TBs, showed that strenuous exercise caused alterations, specifically in the collagen network of the fetlock joint (proximal first phalanx) articular cartilage, including a sharp decrease in hydroxylsypyrindoline cross-linking.

Cu is a component of the enzyme lysyl oxidase required in the protein cross-linking process. Amongst other causes, a dietary Cu deficiency is associated with a failure of capillary endothelial cells to penetrate the distal region of the hypertrophic zone. Hypertrophic chondrocytes secrete: (a) metalloproteinase enzymes including lysyl oxidase, and (b) a basic fibroblast growth factor (bFGF), which stimulates proliferation and migration of endothelial cells and so the angiogenesis of this region in the foetus. The capillary invasion appears to be inhibited by large carbohydrate meals, and the hypertrophic zone then fails to mature.

The primary lesions of retained cartilage may be susceptible to further damage within the joint and secondary lesions of OC, or osteochondritis dissecans, result. Thus, the immature region extends, osteoid does not form normally, so calcification is faulty and the bone is weakened. Subchondral fracture lines may be apparent, loose pieces of tissue appear in the joint (OCD) and synovitis develops. Bone fragments occur particularly at the distal extremity of the tibia (Jeffcott 1991). Secondary osteoarthritis, occurring in the articulations of the cervical spine, causes stenosis of the vertebral canal and signs of ataxia (wobbler syndrome).

There are particular predilection sites of DCP. These are the shoulder and fetlock in the foreleg, the stifle and hock in the hind leg and the cervical spine (Jeffcott 1991). During the suckling phase, the incidence of DOD is slightly greater in colts than in fillies, and certain breeds may be more prone. These include Standardbreds, TBs and Quarter Horses; however, it may be that a closer scrutiny has been paid to these breeds. The pony was thought to be exempt from risk, but the author's own studies in Newmarket (D. Frape unpublished observations) show this not to be so.

Clinical signs may, or may not, be present, but biomechanical stress can precipitate them. Dyschondroplasia may lead to joint stiffness, joint distension (e.g. 'bog spavin'), angular limb deformities and flexural deformity. The first signs occur at various ages: in very young foals, shortly after weaning, or in yearlings and older animals, particularly with the onset of training. However, the peak incidence seems to occur between weaning and the end of December in northern latitudes.

Intake of dietary protein, soluble carbohydrate and energy and effects of exercise

Diets providing excessive amounts of soluble carbohydrate (digestible starch) and total energy [129% of the recommendations of the NRC (1989)], given to foals from 130 days of age, caused widespread lesions of dyschondroplasia (Savage *et al.* 1993a,b). In both of these studies part of the energy increase was achieved with extra

fat and so it may not be concluded that the adverse effects were solely, or necessarily, the result of a raised postprandial insulin response (see below).

Insulin-like growth factor-I (IGF-I) also functions in normal maturation and growth of articular cartilage. Higher plasma IGF-I levels were noted in TB foals given a soluble carbohydrate supplement to pasture, compared with those given a supplement of slowly fermentable carbohydrate and fat. Staniar *et al.* (2001) suggested that the latter supplement may have reduced the risk of DOD. In comparison, Ropp *et al.* (2003) failed to find an effect on growth in Quarter Horse foals, or on their serum IGF-I, when given brome hay with a concentrate containing 103 g fat and 240 g starch per kg cf. 22.1 g fat and 339 g starch per kg. A fat-supplemented diet, cf. a carbohydrate diet, decreased plasma triacylglycerol concentration in Shetland ponies, associated with an increase in plasma lipoprotein lipase activity, whereas a glucose tolerance test led to a massive increase in the insulin response in the fat-supplemented ponies (Schmidt *et al.* 2001) (see below, 'Dietary fats and fibre', for the effects of fat on Ca absorption). Thus, the effects of fat supplements on DOD is unclear.

A dietary protein level of 126% of NRC (1989) recommendations had no significant adverse effect, compared with 100% of those recommendations (Savage *et al.* 1993a). High protein diets do not seem to predispose foals to dyschondroplasia. The author increased the protein allowance and quality of a foal-weaning diet bringing about an increase in growth rate and a reduction in the clinical evidence of physitis, and flexural and angular deformities among successive crops at three TB studs (Frape 1989). Foals weaned at three to four months of age were given this diet *ad libitum* as a coarse mix diluted with 100 g molassed chaff/kg. Glade (1986) postulated that nutritionally induced effects on cartilage growth are mediated by the endocrine system. A single meal initiates insulin secretion and a T_4 response. High plasma concentrations of insulin, resulting from the consumption of large amounts of glucose-yielding carbohydrate, can inhibit growth hormone. The insulin apparently stimulates an early postprandial clearance of T_4 and its conversion to T_3 . Powell *et al.* (2000) found that TBs given only 70% of their daily energy needs failed to produce a T_3 or T_4 response to the consumption of 1.0 kg oats as the morning meal, a response that occurred when the TBs had received adequate amounts of energy daily. Feed intake level affects postprandial hormone responses in addition to that of insulin. The influence of short-term feed restriction may occur through a reduction in the rate of glucose uptake, especially in horses adapted to high roughage.

Glade suggested that DCP has similarities to hypothyroidism (see Chapter 3, Iodine) and the episodic transient postprandial hypothyroidaemia, produced by a high carbohydrate meal, could cause DCP. The *ad libitum* feeding in the author's study (Frape 1989) was intended to exclude large intakes of glucose-yielding carbohydrates during any one hour of the day, so that hyperinsulinaemia would be avoided.

The effects of high energy diets on the abnormal development of joints seem to be independent of their effects on growth rate, but the frequency and severity of lesions

are less where foals are exercised. Such exercise is generally to be recommended, except where severe lesions already exist. With adequate dietary energy, Raub *et al.* (1989) demonstrated that as little as 20 min of medium trot five days per week between 147 and 255 days of age in TB and Quarter Horses was sufficient to increase the radiographic density of the medial side of the third metacarpal and to increase its circumference (the increase in density may simply reflect an increase in circumference). An increase in the breaking strength of the bones should then have been achieved. Several more recent studies confirm the value, and risks, that attend exercise in this context. Bird *et al.* (2000) demonstrated that the introduction of exercise (4 km at 12 m/s to 1 km at 15 m/s) in 24-month-old TB horses increased the rate of proteoglycan synthesis by the chondrocytes in leg joints.

Firth *et al.* (1999) examined the effect of exercise on trabecular thickening (sometimes referred to as osteosclerosis) in the third and radial carpal bones. Exercise increased the density of the subchondral cancellous architecture in regions underlying sites of cartilage degradation. The authors considered that the initiation of localised osteogenesis of cancellous bone was not a consequence of microfractures, but could be purely physiological, occurring without loss of osseous tissue integrity. This hypertrophic stiffening response reduces the shock-absorbing capability, inducing destructive loading of overlying articular cartilage. The critical limits and timing of exercise need to be determined individually for horses.

Angular deformities and growth

Brauer *et al.* (1999) analysed the intracarpal angular deviations of horse legs from radiographs. Horses possess a natural growth-correction mechanism that spontaneously corrects many deformities in foals born with abnormal conformation. The physal growth curve states that, short of pathological limits, cells of the physis that are loaded more heavily grow faster and those loaded less heavily grow more slowly. As the concave side of the physis is the more heavily loaded, the acceleration in growth on that side of the limb tends to lengthen the side until it is no longer concave, or until the load on the physis is balanced along the axis of the limb. This response occurs only where there is dynamic loading, where the pressure is applied and removed intermittently (i.e. with exercise and rest). Static loading retards growth. At the pathological limit (extreme deformity), growth stops and the angulation worsens with continued growth on the convex side of the physis. Correction then requires surgery and or corrective foot care and exercise manipulation (Bramlage 1999).

Restricted dietary protein

There is a tendency for TB and many other breeders to provide weaned foals and yearlings with less dietary protein than is recommended by the NRC (1989). O'Donohue (1991) found that, of the nutrients he measured, dietary protein was the only one for which the daily allowance of short yearlings (towards the end of the first

winter housing period), on 46 Irish stud farms, was on average less than that recommended by the NRC. The effect of this will have been to lower the rate of growth, with the objective of reducing the risk of DOD. There is some evidence that DOD is more prevalent in overweight horses, although from evidence we and others have produced (reviewed by Frape 1989), this may be caused by excessive dietary energy and otherwise badly balanced diets. O'Donohue found that vitamin and mineral supplementation in studs was a 'hit and miss' affair. Supplementary allowances of vitamins A and D ranged from 0 to 18 times the NRC (1989) recommendations. Nevertheless, faster growing horses may still be more prone to osteochondrosis (Sandgren 1993). Yet in the latter Scandinavian study, the faster growing horses were proportionately heavier at birth.

Normal feeding practice and meal frequency

One consequence of rising labour costs has been to decrease the number of feeds per day. If energy-rich feeds are fed in large amounts, say once daily, rather than in smaller amounts in three meals, then there may be an increasing risk of DOD from very large insulin responses. O'Donohue (1991) found that once daily feeding of TB short yearlings was gaining in popularity, that there was a tendency for DOD to be more prevalent where once daily feeding was practised, and that 67% of the 1711 foals he examined in Ireland showed some signs of DOD, although only 11.3% were deemed to need treatment (cf. 10–16% in Scandinavian Standardbreds, Sandgren 1993). Of the treated cases, angular limb deformities and physeal dysplasia together constituted 72.9% of the total. O'Donohue indicated, in agreement with the author's experience in England (D. Frape unpublished observations), that the peak clinical expression of DOD occurred between weaning and the end of December, during the introduction of concentrate feeds. Lesions of OC have been detected between birth and three months of age (Sandgren 1993), so that the stress of weaning may simply exacerbate an existing condition. With increasing labour costs, automated or *ad libitum* feeding systems, to provide more frequent feeds to yarded stock, could be a welcome development, as the author has found (Frape 1989).

Minerals, trace elements and DOD

In Chapter 3, discussing the nutrition of Ca, P and trace elements, it was pointed out that excessive dietary P consistently produces lesions of dyschondroplasia, although clinical signs of NSHP normally do not occur when adequate dietary Ca is provided. Savage *et al.* (1993b) gave foals from 130 days of age a diet providing 388% of NRC (1989) P recommendations, but 100% of the Ca recommendations, and found severe lesions of DCP without clinical signs of NSHP. The incidence of DCP was much greater than in those given 342% of the Ca with 100% of the P recommendations, or in those given 100% of both recommendations. The high P diet would have a lower cation:anion balance (see Chapter 9 and Appendix D) and may therefore have caused some acidosis which would induce bone Ca mobilization. A diet of low

and negative dietary cation–anion difference (DCAD) causes chronic metabolic acidosis and an elevation of plasma parathyroid hormone (PTH) that stimulates the conversion of 25-(OH)D₃ to 1,25-(OH)₂D₃ in the kidney. This regulates the active transport of Ca across the intestine. If growing horses are receiving adequate dietary Ca, but a diet of low DCAD, then the stimulation to intestinal absorption of Ca may compensate for the increased urinary Ca loss. Surprisingly, this has led in growing horses to a greater positive Ca balance than occurs with a diet providing the same adequate amounts of Ca, but a high DCAD (Cooper *et al.* 1999b). The importance of adequate dietary Ca is paramount.

Dietary fat and fibre

Hoffman *et al.* (1999) gave mares and their foals up to 12 months of age supplements providing 150 g protein/kg, but differing in fat and fibre contents. Post-weaning, the foals received 1.0 kg rising 1.4 kg supplement twice daily. Bone mineral content was lower in weanlings and yearlings given the supplement containing per kg 10.1 g Ca, 412 g NDF and 104 g fat rather than the supplement containing 7.7 g Ca, 153 g NDF and 24 g fat, as adjuncts to a blue grass/white clover pasture, or mixed grass/legume hay in winter. There was a tendency for a greater incidence of angular deformities and flexural deformities during June and angular deformities during January in foals given the supplement containing more fibre and fat. The authors suggested that fats forming calcium soaps and fibre capturing cations in the gut, may have adversely limited the supply of bone mineral.

Dietary trace elements

Pregnant mare and foal diets containing less than 10 mg Cu/kg may cause DOD and flexural deformity. Lysyl oxidase is a Cu-containing enzyme required for the cross-linking of protein chains in elastin and collagen of cartilage through the lysine residues, by oxidizing the ε-amino group. Failure of this function disrupts normal bone cartilage development. Dietary Cu supplements for the pregnant mare and concentrations of up to 30–45 mg Cu/kg in the weanling's feed may decrease the incidence of DCP and physitis in growing horses. These levels of Cu are absolutely safe and well below any toxic threshold, as the horse has a considerable resistance to chronic Cu toxicity. Cu supplementation of the lactating mare has no measurable effect on the Cu content of the milk, which is quite low.

Glucosamine, methyl sulphonyl methane and chondroitin sulphate supplementation

Glucosamine

The rate-limiting step in the glycosylation of many proteins is probably the formation of glucosamine-6-phosphate catalysed by the enzyme glucosamine synthetase.

Glucosamine, modified as N-acetylglucosamine, constitutes half the molecular composition of hyaluronic acid. By isomerisation, glucosamine can be converted to galactosamine and so can contribute half the molecular composition of chondroitin sulphate. Glucosamine stimulates both proteoglycan and collagen synthesis, so that it may help regeneration of cartilage matrix and the proteoglycan synthesis may reduce inflammation within damaged joints. Nevertheless, a large proportion of the absorbed glucosamine molecule is modified by the liver and utilised for energy production, hence the most effective dose may be high. Chondroitin sulphate supplementation is also probably effective, but evidence is scarce (Platt 2001).

Increased proteolytic enzyme activity is a major factor responsible for degradation of extracellular matrix of articular cartilage. Glucosamine may increase synthetic activity of chondrocytes. The addition of glucosamine to equine articular cartilage discs treated *in vitro* with lipopolysaccharide, induced decreased gelatinase and collagenase activity and inhibited nitric oxide production, so inhibiting prostaglandin release (Fenton *et al.* 1999), indicating a complexity to the action of glucosamine. As well as glucosamine, mannosamine has inhibited equine 'knee' cartilage degradation, *in vitro*, in the presence of lipopolysaccharide, although mannosamine has been effective at less than half the concentration of glucosamine (Mello *et al.* 2001). Electron microscopic examination in humans shows that the consumption of a glucosamine supplement, cf. placebo, may improve physical performance and knee cartilage health, reversing cartilage degeneration.

Methyl sulphonyl methane (MSM)

Anecdotal evidence indicates that MSM may relieve inflammation and pain due to arthritis and muscular disorders, although there has been little equine evidence to show that it is absorbed and utilised. Pratt *et al.* (2001) demonstrated in adult horses that 55% of S of MSM origin was absorbed and large amounts accumulated in the blood. The technique employed indicated that the MSM was probably absorbed intact (see also Chapter 5 and 'Control of growth abnormalities', Chapter 7).

Summary of control of DOD

In the rearing of foals:

- Avoid large meals rich in glucose-yielding carbohydrate, but ensure the protein requirements are met with balanced amino acids.
- Provide adequate dietary Cu, but with a trace-element balance.
- Provide adequate dietary Ca with a correct Ca:P ratio.
- Provide adequate exercise daily.
- Avoid breeding from stallions that are genetically predisposed to produce dyschondroplasia-affected foals.
- In studs where there are risks of dyschondroplasia, the foals should be given no more than the recommended NRC rates of DE intake.

- Where clinical DOD occurs, the energy intake of foals should be decreased and the joint trauma minimized by restricting the foal to hand-walking exercise until signs disappear.
- In studs where good labour is scarce, consider the introduction of automated feeding to avoid large meals, so long as careful daily observation, by experienced persons, of each foal is not compromised and so long as obesity is avoided.

STUDY QUESTIONS

- (1) What factors are important in selecting a suitable pasture for weaned foals?
- (2) What action should be taken with a foal presenting mild signs of orthopaedic disease, or with moderate signs, at an early age, or postweaning?

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Chapter 9

Feeding for Performance and the Metabolism of Nutrients During Exercise

But if you intend the next day to give him an heat (to which I now bend mine aim) you shall then only give him a quart of sweet oats and as soon as they are eaten, put on his bridle, and tie up his head, not forgetting all by ceremonies before declared.

T. De Gray 1639

Feed costs are major outgoings in the breeding, training and use of sport horses. Corbally (1995) found that in the Irish sport-horse-breeding industry feed was the second largest cost after labour, representing 16% of the total cost when the costs attributable to amortization of buildings, and brood mares and the current expenditure on labour, stud, veterinary surgeons, schooling, bedding, farrier, showing, tack and equipment and registration were also included. At equestrian sports centres feed was the second largest cost, representing 19.4% of the total. It is therefore important to the economy of the industry that feed has the optimum composition and is used judiciously. This chapter, however, considers the principles underlying the feeding and metabolism of nutrients, so that composition of feed and methods of its provision may be manipulated to optimize performance and minimize the risks to the horse and rider. For brevity the discussion is restricted to the relationship of performance to the functioning of tissue cells and their nutrition.

WORK AND ENERGY EXPENDITURE

A major role of feed for working horses is the conversion of the chemical energy of feed into locomotion at speeds varying from 160 to over 900 m/min (6–35 mph) for distances varying from 1–150 km or more. This enormous range may superficially lead to equal fatigue in fit horses, but quite different processes of nutrition physiology are involved at the two extremes of distance and speed. At the one extreme a flat race of 6–8 furlongs (1.2–1.6 km) would *theoretically* increase the day's energy needs by a mere 4% (Plate 9.1), a barely perceptible effect, whereas at the other they would be increased by five- to sixfold. Training regimes recognize both this and the different responses of breeds to contrasting forms of work. These distinctive training procedures induce profound and dissimilar physiological changes in the attainment of fitness. Diets must be formulated to conform with these distinct needs, but appetite may flag in the process. First, an adequate and optimum nutrition for a



Plate 9.1 Rathgorman, ridden by Chris Bell, cantering 8 furlongs (1.6 km) on the all-weather track at Dunkswick, West Yorkshire, watched by trainer Michael W. Dickinson. As part of the training for National Hunt racing, the horses canter five days per week. If a horse races every 7–21 days it has an easy canter on the day before and for several days after a race. A regular work programme is maintained during the racing season but with some variety in location, scenery and type of work.

particular purpose implies an optimum supply of nutrients to each tissue and cell and the efficient disposal of waste products.

In sprint races, horses obtain much of their muscular energy from anaerobic pathways of respiration, whereas in extended work, such as endurance competitions, energy is derived almost exclusively through aerobic pathways. (Anaerobic respiration is the breakdown of organic nutrients in the absence of oxygen but with the release of energy captured by ATP.) A day's hunting, with episodes of hill climbing, cantering and jumping, and periods of waiting and walking, combine both processes and rates of energy expenditure. The expenditure by a hunter carrying a heavy huntsman during a long day exceeds the average daily consumption of feed energy by several-fold. The processes of energy metabolism are summarized here, as a greater understanding of them should allow more rational feeding, and it should foster comprehension of each future development in the feeding of working horses.

What is work?

Before proceeding further it may be appropriate here to review the meaning of the terms 'force', 'power', 'work' and related terms and see how they have been applied to measurable and practical work of working horses. If a horse was a smooth ball on

Table 9.1 Mean draught load, distance travelled, work done and net energy expended, in addition to maintenance, during a six-hour day, by five Chilean draught horses ploughing a field (Pérez *et al.* 1996). The horses were exercised under submaximal conditions, although there was a significant increase in blood cortisol concentration.

Live weight (kg)	547
Force applied, or draught load ¹ (N)	905
Distance travelled (km)	13.6
Speed (m/s) ²	0.93
Power developed (kJ/s or kW, i.e. kilowatts)	0.83
Work done in pulling above maintenance (kJ/horse)	12276
Work done over six-hour day (kJ/kg BW ^{0.75}) ³	108
Estimated net energy for work, including horse moving itself (kJ/day) ⁴	130584
Estimated net energy used for work, including horse movement (kJ/kg BW ^{0.75})	1153
Estimated gross efficiency of work, including horse movement, but excluding maintenance (%)	9.4
Net energy for work expressed as multiple of maintenance	2.24

¹Mean force exerted by the five horses was equivalent to 17.7% of their body weight, or a draught power of 780 watts, Js⁻¹.

²18.5 hours would be required to plough 1 ha.

³This is greater than that achieved by oxen, as the horse requires about two-thirds of the time to achieve a similar task.

⁴Energy requirement for this work of six-hour is about 125% of that required for two-hour medium trot daily including rider and maintenance.

a frictionless horizontal surface, no energy would be required for it to continue moving at a constant speed in a straight line. Fortunately, for both the horse and feed manufacturer, all movement of the horse involves friction, both in the horse's motion (an assumed value of 2J to move 1 kg BW 1 m horizontally) and in anything the horse may be pulling. It requires force to overcome the friction, and the force, acting over a given distance, equals the work output. If hill climbs intervene, the force of gravity has to be overcome as well. The extra energy required above maintenance is:

$$2J[BW \text{ (kg)} \times \text{horizontal distance (m)}] + \frac{\text{work done}}{0.298}$$

where 0.298 represents the ratio of work done pulling/net energy used (cf. gross efficiency, Table 9.1). Force is measured in newtons (N), joules per metre (J m⁻¹) or kilogramme metres per second squared (kg ms⁻²). Work output is measured in newtons, N, for the distance over which the force is applied, expressed as joules, Nm = J.

The working horse

One of the most traditional forms of work for a horse must be that of pulling a plough. Several groups of workers have accurately measured the work of ploughing. The power output, measured in watts (Js⁻¹) per unit live weight, in ploughing 4

hours/day was similar between two Jersey crossbred oxen (323 kg BW per head) and four heavy donkeys (170 kg BW per head). Donkeys tended to plough at a slower pace than the oxen (Nengomasha *et al.* 1999). Pérez *et al.* (1996) reported on five Chilean crossbred draught horses weighing 547 kg, pulling a mould-board plough, cutting a furrow of depth and width 12.6 and 22.3 cm, respectively. Of course, the measured performance is influenced by the soil characteristics, but the values they obtained (Table 9.1) agree well with those obtained by others and are informative to the nutritionist.

The riding horse

Energy expenditure and work type

Endurance work is at the rate of 154–224 m/min (5.75–8.35 mph) over variable slopes, whereas flat racing is at about 940 m/min (35 mph). For comparison, the stage-coach horse, pulling a heavy load, covered about 40 km (25 miles) at speeds similar to those required of endurance horses – 200–214 m/min (7.5–8 mph). Strenuous effort tends to depress appetite, so recovery from extreme effort requires several days for reserves to be replenished. The data in Fig. 6.4, p. 190, and Table 6.8, p. 211, show that the capacity of larger horses for feed is proportionally less than that of smaller horses, so that after undertaking strenuous work they may require a longer period for recovery, especially if it is assumed that the weight of the rider is proportional to the weight of the horse. The information in Table 6.3, p. 201, is based on experimental results, and, although there has been some extrapolation, it does indicate greater demands for feed energy than the theoretical estimates for endurance work given in Table 6.8. The NRC estimates in Table 6.8, however, seem to exceed the INRA estimates; see Table 6.23.)

Blaxter (1962) calculated that the energy expenditure for vertical effort in horses, in addition to any horizontal effort, amounted to 17 times that expended in horizontal movement, above the costs of energy metabolism at rest. A 400 kg horse is calculated to expend 0.67 kJ/m in horizontal work and 11.4 kJ/m vertically. Exercise over uneven and hilly ground can therefore be much more arduous than that on the flat. Sprint work on the flat must be considered a special case, as it is almost entirely confined to young horses which may still be growing. Extended work, for which there is somewhat more published experimental evidence, is generally undertaken by older horses.

Work, especially, entails an increase in nutrient supply to the muscles, and in converting glucose or free fatty acids to high-energy phosphate compounds – ATP and CP or phosphocreatine (PCr) – which the muscles use as an immediate source of energy. There is an increase in the rate of production of waste products, more particularly carbon dioxide and heat. The supply of nutrients and the effective disposal of wastes entail large physiological adjustments, which training seeks to encourage. The most critical feeds for work are those that provide energy, water and electrolytes. Electrolytes are those elements that in solution carry an electri-

cal charge. They include sodium, potassium, magnesium, calcium, chloride and phosphate.

ENERGY SUBSTRATES AND THEIR EXPENDITURE

Most potential energy for muscular work is absorbed from the intestinal tract as glucose, VFAs (acetic, propionic, butyric and smaller quantities of related acids), longer-chain fatty acids, neutral fats and amino acids. Absorbed glucose, propionate and glucogenic amino acids are potential sources of blood glucose and of liver glycogen, a storage starch. Absorbed long-chain fatty acids, neutral fats, ketogenic amino acids and particularly acetate and butyrate are potential sources of blood fats and fatty acids, storage fat and acetyl-CoA. Blood glucose and its precursors are also, of course, potential sources of body fat through the key substance acetyl-CoA.

ATP and CP (PCr) formation and use

The liver, while storing energy in the form of glycogen and fat (and also as protein), serves the vital role of maintaining normal levels of blood glucose through the breakdown and re-storage of the glycogen. Muscle cells also store glycogen and form high-energy phosphate compounds – CP and ATP – necessary for muscular relaxation and contraction, by drawing on blood glucose and fatty acids as fuels. The complete release of chemical energy from them requires a supply of oxygen (O_2) reaching the muscle cells through the arteries. However, an immediate and rapid release of energy, particularly important in short sprint races, can be achieved through the process of glycolysis, in which glucose is broken down to pyruvate in the muscle cell without the consumption of O_2 , and also by the release of energy from previously stored ATP and CP. The further and complete breakdown of pyruvate and of fatty acids demands the presence of O_2 and this process takes place exclusively in the mitochondria, through the agency of what are known as β -oxidation of fatty acids and the TCA cycle.

ATP resynthesis

Energy for muscle contraction is derived from CP (PCr) and ATP which are formed from the energy derived during the combustion of glucose and fatty acids. TBs possess a high proportion of PCr-rich fast-twitch fibres in skeletal muscles (Harris & Hultman 1992a), so that loss of adenine nucleotides (AN) during intense exercise is greater than in man (see 'Ammonia and the alanine vehicle', this chapter). After repeated gallops, a 50% loss of ATP was recorded (Harris *et al.* 1991c; Sewell & Harris 1991; Sewell *et al.* 1992a), associated with fatigue and a decrease in running speed (Harris *et al.* 1991b), and with lower glycolytic rates and muscle (ATP)/(ADP). The accumulation of ADP stimulates AN degradation to inosine monophosphate (IMP) with an increase in plasma NH_3 concentration. There is a critical pH below which ADP rephosphorylation declines (see 'Ammonia and the

alanine vehicle', this chapter), PCr acting both as an intracellular buffer and a reservoir for this rephosphorylation. Supplementation of human subjects with *creatine monohydrate* has increased muscle total creatine and PCr contents (Harris *et al.* 1992). The energy reserves for this synthesis depend upon training, diet and the inherent characteristics of the horse. The slow rate of ATP resynthesis, when supported by free fatty acid (FFA) oxidation, means that inadequate glycogen storage in active muscle fibres causes early fatigue despite an abundance of fatty acids.

TCA cycle

The operation of the TCA cycle in the mitochondria requires the diffusion of O_2 to these organelles and the removal of CO_2 to the blood. During light work, this process proceeds smoothly, and with each turn of the cycle one unit of oxaloacetate, which is required for the subsequent metabolism of acetyl-CoA in the presence of O_2 , is produced. However, when larger quantities of oxaloacetate are present, the metabolism of acetyl-CoA is likely to proceed more rapidly. When larger quantities of fatty acids are dissimilated to acetyl-CoA, the cycle must turn at an accelerating rate. This is also achieved by the provision of extra quantities of oxaloacetate from outside the mitochondrion. There must, therefore, be adequate quantities of pyruvate requiring an ample supply of glucose, lactate, glucogenic amino acids or even glycerol. During extended work, the trained healthy horse finds no difficulty in breaking down fatty acids to CO_2 , as sufficient O_2 is taken in by normal respiration. In fact, such work leads to an accumulation of glycerol, signifying that it is not called on to form pyruvate units in any great quantities. If the utilization of fatty acids was interrupted, this would probably be expressed as a retarded metabolism of acetyl-CoA and there would be a build-up of blood ketones (acetoacetate and 3-hydroxybutyrate).

Work in Newmarket (Frape *et al.* 1979) has shown that these ketones accumulate in the plasma only after work stops (Fig. 9.1), possibly implying that no bottleneck occurs to the complete combustion of fats. This post-exercise rise in plasma ketones probably reflects the redistribution of blood from muscle to adipose tissue from which NEFAs will then be flushed, rapidly increasing the plasma concentration of their metabolites, owing to a reduced need for their cellular consumption. Hyperlipidaemia, recognized in starving horses, reflects a blocking of fat metabolism in animals relying overwhelmingly on residual fat stores, in the relative absence of carbohydrate substrates. The activity of enzymes required in this fat breakdown may also be depleted in an associated deficiency of dietary protein. Figure 9.2 gives a brief account of the pathways by which energy sources are metabolized in horse muscle cells and liver.

Glycogen, glucose, FFAs and VFAs

The amounts of muscle and liver glycogen stored at any one time is variable, as reserves are considerably depleted during extended exercise and are replenished

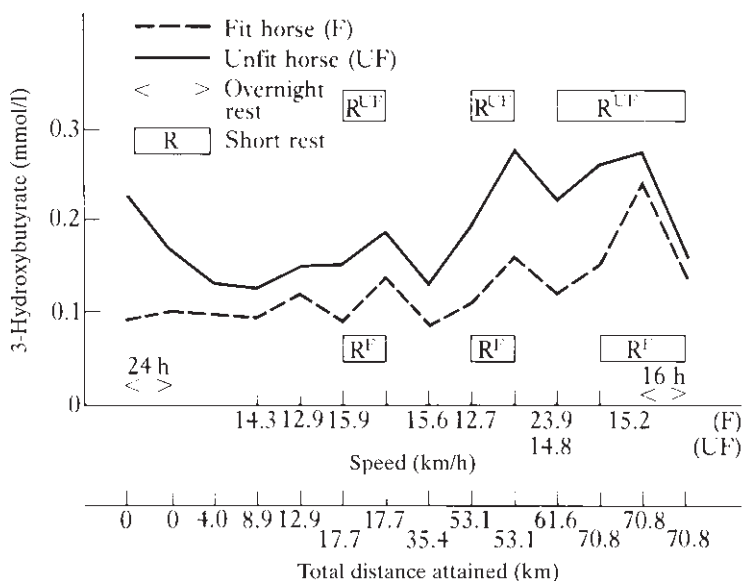


Fig. 9.1 Effect of rest periods during an endurance ride on plasma 3-hydroxybutyrate in a fit and an unfit horse (Frape *et al.* 1979).

only over several meals. Nevertheless, the author calculates that, on average, liver reserves of glycogen are 5–10% of those in skeletal muscle, making assumptions concerning muscle mass and that the liver represents 1.5% of body weight. Glycogenolysis, or the breakdown of glycogen, continues following the completion of intensive exercise. Recovery of these glycogen stores is promoted by giving a meal of 1–2 kg cereal 1.5 hours following exercise, or by the i.v. infusion of 6 g glucose/kg BW, 30 min postexercise. This may have implications for horses competing on successive days. On the other hand, in trained horses, glycogen stores do not limit sprint performance, as training can increase glycogen capacity of limb muscles by 39% (Guy & Snow 1977a).

With increasing rates of energy expenditure, the preference for glucose as a substrate increases, although the proportions of glucose and FFA used change rapidly with distance. Glycogen utilization rates of 2.68 and 1.06 mmol glucosyl units/kg dry muscle/s with total consumptions of 27.3 and 32.5% of the initial store were caused, respectively, by 800 and 2000 m gallops (Harris *et al.* 1987). This suggests that over the greater distance, at a somewhat lower velocity (aerobic) combustion of FFA increases considerably as a proportion of total energy expenditure.

In addition to FFA derived from the hydrolysis of fat stored in adipocytes, VFAs absorbed from the hind-gut are an important energy source. The contribution of acetate to oxidation in the hind limb was reduced from 32% in horses given roughage to 21% in those given a diet with an oats:roughage ratio of 0.52:0.48 (Pethick *et al.* 1993). This may be one reason why higher roughage intakes are recommended for endurance horses (see also 'Sweat and dehydration', this chapter).

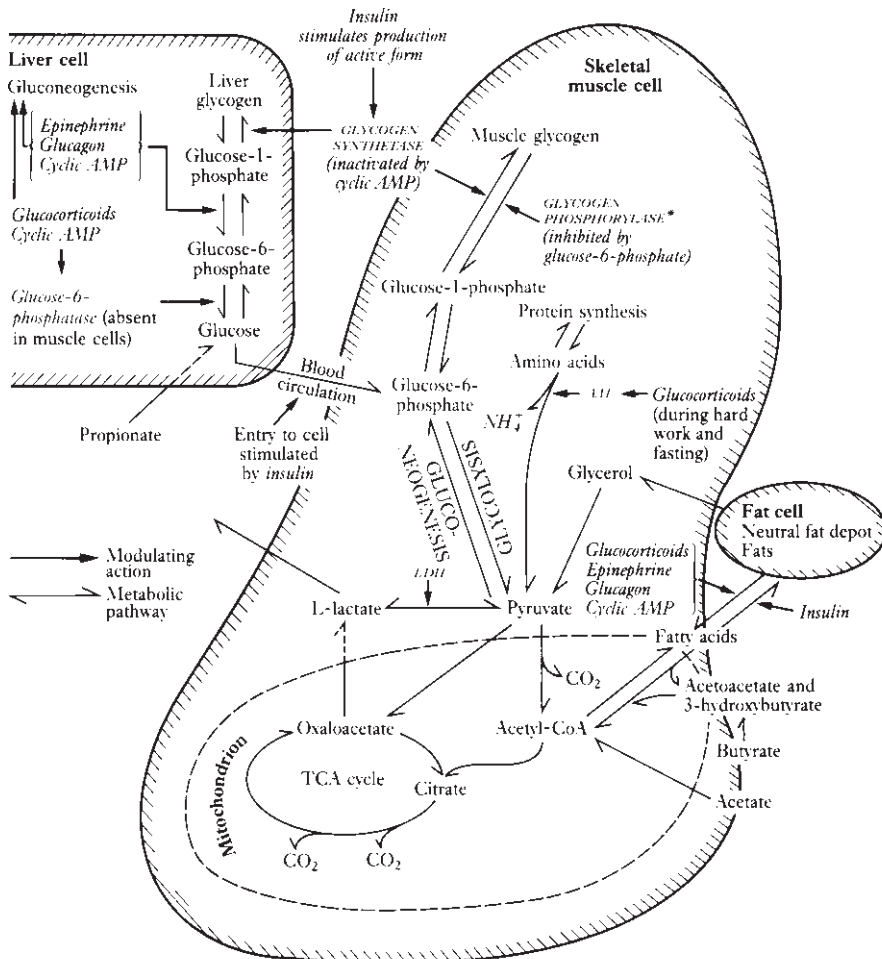


Fig. 9.2 Major pathways of energy metabolism in muscle and liver cells and their modulation by the endocrine system (intermediate steps have been excluded). Glycolysis: the oxidation of glycogen or glucose to pyruvate and lactate. Gluconeogenesis: the formation of glucose or glycogen from non-carbohydrate sources. The principal substrates for this are glucogenic amino acids, lactate, pyruvate, glycerol and, to a lesser extent, propionate.

*Bursts of muscular action with the secretion of glucagon and epinephrine and nervous stimulation of the release of Ca^{2+} initiate the formation of cyclic adenosine monophosphate (AMP) and then the phosphorylation and activation of glycogen phosphorylase.

Pyruvate–lactate: the oxygen debt

The anaerobic production of energy by glycolysis would soon be halted by an excessive accumulation of pyruvate. A cunning mechanism has therefore evolved in which pyruvate is reversibly converted to lactate as an intermediate ‘waste’ product. This reduction even more importantly oxidizes reduced NAD^+ , essential for triggering an important glycolytic step. Therefore, in sprint racing, lactate diffuses into the

blood from the muscle cell and there accumulates until sufficient O_2 is available for its hepatic conversion to pyruvate. This mechanism allows more energy to be obtained by glycolysis than would otherwise be possible. As the O_2 debt has eventually to be repaid, the yield of energy per unit volume of O_2 consumption, owing to anaerobic processes, is only half that achieved by aerobic processes. However, during recovery from sprint races, O_2 is abundant so that the advantage of the rapid availability of chemical energy outweighs the minor disadvantage of interest payment.

Lactic acid threshold

Whether sufficient O_2 is present in muscle cells depends on the speed and the extent of training of the horse. The anaerobic lactic-acid system in unconditioned TBs is probably untaxed until work rates exceed around 600 m/min (22 mph) on the flat (Williamson 1974). The anaerobic threshold of Standardbred trotters is said to be 300–400 m/min (11–15 mph) on the flat (Milne *et al.* 1976), although a striking increase in blood lactate of Standardbred trotters was not observed until speeds exceeded 600 m/min (Lindholm 1974), or 684–750 m/min (25–28 mph). Lindholm & Saltin (1974) and Williamson (1974) recorded similar blood responses in these breeds undertaking the same types of exercise.

The threshold of exercise-induced lactate accumulation may be taken to be 4 mmol lactate/l lactate in the plasma, and the velocity for a particular horse at which this occurs is termed $\dot{V}_{LA4}$. Anaerobic metabolism provides approximately 30% of immediate energy needs of Standardbreds running at racing speeds. Conditioning by repeated high-intensity exercise (92% $\dot{V}O_{2max}$), produced: increased muscle glycogen concentration before exercise; a higher $\dot{V}_{LA4}$; increases in $\dot{V}O_{2max}$ and in speed at which $\dot{V}O_{2max}$ was achieved; speed at 115% of $\dot{V}O_{2max}$; and an increase in the duration at which 115% of $\dot{V}O_{2max}$ was maintained by Standardbreds (Hinchcliff *et al.* 2002). Oxygen consumption was greater and accumulated oxygen deficit was higher than it was before conditioning. O_2 deficit is defined by the O_2 equivalent of the difference between ATP supplied oxidatively from pulmonary $\dot{V}O_2$ and the ATP utilised in the exercising muscle (see Glossary). (NB: TBs, as a breed, have a higher mean $\dot{V}O_{2max}$ and a greater ability to produce lactate than the Standardbred. Moreover, Ronéus *et al.* (1999) concluded that plasma lactate and ammonia concentrations in Standardbred trotters after racing were unrelated to current race performance.) Thus conditioning caused an increase in both aerobic and anaerobic capacity.

However, there were lower muscle lactate concentrations after high-speed exercise, but similar blood lactate concentrations during exercise at $\dot{V}O_{2max}$ and recovery, so that conditioning may enhance the rate of efflux of lactate from muscle (high-intensity conditioning causes an increase in muscle LDH, see Glossary). The lower muscle lactate and a raised intramuscular pH may well have reduced the energetic cost of muscular function. If so the increase in O_2 deficit could be explained by this in the absence of demonstrated increases in the net energetic contributions of

anaerobic glycolysis and/or phosphagen and O₂ stores (Poole *et al.* 2002). The authors concluded that high-intensity exercise resulted in reduced reliance on anaerobic metabolism at similar absolute work intensities.

This explanation is supported by the observation of other investigators that conditioning results in a reduction in respiratory exchange ratio (rate of CO₂ output/rate of O₂ uptake) during intense exercise (NB: A decrease in the ratio occurs when there is an increase in the proportion of total energy derived from fat oxidation). However, associated with a greater work output the conditioning increased the calculated maximal O₂ deficit in horses. Horses with the greatest anaerobic capacity that can generate the largest O₂ deficit will have a significant race advantage.

In a study by workers at the University of Glasgow (Snow & MacKenzie 1977b), cantering for up to 22km caused no stress on nutrient reserves in the blood in that, immediately after the exercise, blood glucose, glycerol and FFAs, as well as pH, rose slightly, which indicated an adequate rate of their mobilization and a sufficient irrigation of muscle cells with O₂. Snow & MacKenzie (1977a) found that at a maximum work rate of 864m/min (32mph) over 3 × 600m, with two 5min intervals between the gallops, anaerobic respiration was necessary in that blood glucose, glycerol, FFA and lactate were all elevated and there was a fall in pH. The increased fund of blood glucose was associated with a considerable rise in adrenocorticosteroid secretion. Training for ten weeks before the gallops caused a further increase in blood glycerol and lactate with smaller changes in FFA and pH than was the case with the untrained horses. As corticosteroid secretion was even greater after training, an explanation may be proffered that training confers a more efficient mobilization of reserve fat, and both it and glucose are used to a greater extent by trained horses, resulting in faster times. The lesser fall in blood pH indicates that training also leads to better ventilation and oxygenation of the muscles.

TRAINING METHODS

Muscle type and breed

Long-distance riding requires a preponderance of muscle fibres capable of slow contraction but resistant to fatigue, whereas sprinting ability demands the presence of a higher proportion of fast-contracting fibres that also happen to be readily fatigued. These fast-contracting fibres are categorized as having myosin with high ATPase activity at pH 9.4 and a high glycolytic activity (type II fibres). They are subdivided into fast-twitch, low oxidative (FT) and fast-twitch, high oxidative (FTH) fibres. Fibres with low myosin-ATPase activity (type I fibres) are present in relatively higher proportions in the skeletal muscles of horses more suited to long-distance riding. These are known as slow-twitch, high oxidative fibres (ST). Standardbred trotters older than four years have 54% FTH, 22% FT and 24% ST fibres in the gluteus medius muscle (Lindholm & Piehl 1974). All muscles have about the same

ratio of low oxidative to high oxidative fibres, but a mixture of anaerobic and aerobic training increases the proportion of FTH fibres and decreases the proportion of ST and possibly also of FT fibres. Nevertheless, Dingboom *et al.* (2002) detected a change with time in fibre type of locomotory muscles that was uninfluenced by exercise in Dutch warmblood foals during their first year postpartum.

The purpose of training is to modify muscular action and indeed the whole metabolism of the horse so that it functions at maximum efficiency with minimum fatigue at the speed and over the distance at which the sights are set. Courouc   *et al.* (2002) demonstrated in French trotters that $\dot{V}_{LA2}$ and $\dot{V}_{LA4}$ and the corresponding heart rates HR_2 and HR_4 , and the velocity for a heart rate of 200 beats/min ($\dot{V}_{200}$), all improved with age between one and six years. Moreover, $\dot{V}_2$, $\dot{V}_4$, and $\dot{V}_{200}$ increased with training. This evidence indicates more efficient oxygen transport and aerobic energy production. HR at a given blood lactate content may be improving with age, owing to an increase in the stroke volume of the heart and an increase in the arteriovenous oxygen content difference, brought about by a reduction in the mixed venous blood oxygen content. A low $\dot{V}_{200}$ may frequently indicate an underlying orthopaedic disease, according to several groups of workers.

During exercise, feed energy is converted into work and Table 6.8 gives the approximate amounts of feed energy required by horses of different weights undertaking work of a variety of intensities. In all but one instance, the work covers a period of 1 hour, although it is appreciated that the most strenuous effort could not be sustained continuously for such a period. Nevertheless, the method allows comparisons of different degrees of effort. On the one hand, walking creates practically no further demand on the requirements for maintenance, whereas at the other extreme, racing – and more particularly lengthy endurance work – creates an energy demand that exceeds the capacity of the horse for the immediate replenishment through feeding of the losses. Glycogen and fat reserves are therefore heavily drawn upon.

Muscle hypertrophy

Adaptation to hard muscular work entails changes not only in the blood vascular system but also in skeletal muscle. Hypertrophy of muscle occurs during training, the extent of which varies with the breed of horse and with the type of work. This change is reflected in an increase in apparent nitrogen (N) retention in Quarter Horses (Freeman *et al.* 1988). Losses of N in sweat were not measured, and part of the apparent retention may be accounted for by this loss. The increased retention of N appeared to continue for a month of rest following the exercise period, indicating a higher maintenance requirement for protein in horses that have been in training.

Training effects

As the intensity of training increases, the demand for energy release by way of a particular metabolic pathway rises, so increasing the need for the appropriate enzymes to catalyse the reactions. Sprint racing imposes greater demands on

anaerobic metabolism and longer races call predominantly on aerobic processes. The latter implies a more intense use of the TCA cycle in the mitochondria. Training for sustained work is therefore seen to bring about an increase in the number of these cellular organelles and their associated enzymes. Training for sprint racing, however, increases glycolytic activity and a twofold increase in the activity of the enzymes aldolase (a key enzyme in the glycolytic pathway) and ALT has been observed. The latter promotes the formation of the amino acid alanine (see 'Ammonia and the alanine vehicle', this chapter, for a function of this amino acid) from pyruvate so lessening the conversion to lactate and therefore probably lessening fatigue by moderating a fall in pH.

Exercise induces a transient rise in extracellular K^+ concentration, owing to a continuous loss of K^+ from contracting muscles. This loss affects the membrane potential that may contribute to muscular fatigue. Re-uptake of K^+ by the cell is achieved with the action of the Na^+-K^+ pump ($Na^+-K^+-ATPase$). Suwannachot *et al.* (2001) demonstrated that short-sprint training of Dutch warmblood foals for five months induced an increase of the $Na^+-K^+-ATPase$ concentration in *gluteus medius* and *semitendinosus* muscles. The effect persisted during six months of detraining. These metabolic responses reveal ways in which training can influence specific aspects of muscle function and ultimately performance (see also 'Sodium bicarbonate', this chapter, and 'Dehydration and potassium status', Chapter 11).

MUSCLE ENERGY RESERVES AND FEEDING BEFORE EXERCISE

Hormonal effects

A normal meal of grain leads to an elevation in plasma glucose and insulin concentrations. Insulin elevation can last for a period of up to eight hours. It is an anabolic hormone promoting glucose and fat storage, whereas exercise requires the mobilization of energy reserves for combustion. An intense exercise bout 2–6 hours after a meal, is associated with decreased FFA or NEFA availability, leading to a rapid fall in blood glucose concentration, as the contribution of fat to energy expenditure is decreased. For example, Lawrence *et al.* (1993) fasted Standardbred horses overnight and then gave them no grain (controls) or 1, 2 or 3 kg maize grain 2.5–3 hours before a warm-up and intense exercise over 1600 m at a heart rate of 206 bpm. The controls maintained steady plasma glucose concentrations, whereas plasma glucose declined in those given any grain, a response similar to that observed by others, where horses were fed three or four hours before exercise.

Plasma FFAs were initially higher in the controls, but FFA declined in these horses during the intense exercise. In the grain-fed horses plasma FFA remained steady during exercise, but the concentration was always less than in the controls. The effect of a meal on repeated bouts of intense exercise during a day probably depends on the size of the meal, timing, exercise intensity etc. Lawrence *et al.* (1995) found that a meal neither improved nor impaired the performance of repeat exer-

cise bouts. The reason for this may be that intense exercise stimulates epinephrine and norepinephrine (adrenaline and noradrenaline) secretion which overrides the effect of insulin on glucose and fat metabolism, so that fat mobilization and release of NEFA into the blood would not be impaired during second, third etc. bouts of intense exercise (see also 'Feeding before endurance rides', this chapter.)

Blood distribution effects

(Also see 'Exercise warm-up', 'The vascular and respiratory systems', this chapter, and Fig. 9.7.) Experiments with ponies (Duren *et al.* 1992) indicated that exercise of 7.8m/s on a 6.3° incline at 75% of heart rate maximum for 30 min, 1.4 hours after feeding, led to higher GI tract and skeletal muscle blood flows compared with those in fasted ponies. There was an increase in heart rate, cardiac output, stroke volume and arterial blood pressure, whereas accommodation may not have been possible with more intense work. This work is therefore normally delayed for five to eight hours after feeding, although the optimum time interval is influenced by the dietary proportion subject to fermentation and type of activity.

Glycogen use and muscle type

The accelerated use of glucose is accommodated by a stimulation of glycogen deposition in the muscles of adequately fed trained horses. This training can increase the glycogen capacity of TB muscles by a third. Blood glucose cannot, however, be maintained indefinitely during work, and endurance rides of up to 150km result in a gradual decline and an exhaustion of muscle glycogen. In one 80km (50 mile) ride, blood glucose on average fell by 40%, whereas FFA rose eightfold (Hall *et al.* 1982). In trained horses, blood glucose concentration is not affected by endurance rides of 50km (31 miles), but is decreased 23% during a ride of 100km (62 miles) (Essén-Gustavsson *et al.* 1984).

The net loss of muscle glycogen is extremely small in light work. Lindholm (1974) observed that Standardbred trotters, for instance, lost 0.3mmol glycogen/(kg × min) when trotting at 300m/min (11mph), whereas trotting at a maximum rate (750m/min or 28mph) led to a loss of 14mmol glycogen/(kg × min). Three aggregate minutes of maximal trotting were proved to cause a 48% decrease in muscle glycogen, but the decrement was not equally distributed among the fibre types. Maximal work causes a striking depletion of ST fibres in addition to a loss from the other two types. On the other hand, slow trotting leads to a gradual depletion of ST fibres after which the FTH fibres become active and depleted. Thus, there seems to be a preferential fibre recruitment with increasing speed and duration.

Glycogen 'loading'

The stimulation of glycogen accretion has been considered from both safety and efficacy aspects. Starchy diets and regular exercise can increase muscle glycogen, but this increase is not maintained if the starch content of the diet is subsequently

reduced. Harris & Hultman (1992a) found no difference between diets insofar as loading is concerned, although loading is probably more effective during rest on a high carbohydrate rather than a high-fat diet following intense exercise that depletes type II muscle fibres (fast twitch) of glycogen (Pagan *et al.* 1987a). By comparison with intense exercise, aerobic work is ineffective and glycogen loading may cause poorer performance (Topliff *et al.* 1985, 1987; Pagan *et al.* 1987a) and an increased risk of exertional rhabdomyolysis ('tying-up'), according to a widely held view. This condition may not, however, be associated with either lactic acidosis, or excessive dietary carbohydrate. Hodgson (1993) speculates that horses predisposed to tying-up exhibit a temporary failure in the control of intracellular $[Ca^{2+}]$.

A glycogen storage disorder, described as polysaccharide storage myopathy (PSSM), is a condition in which there is high glycogen and/or abnormal complex amylase-resistant polysaccharide deposits. It has been recognized in Quarter Horses and is considered to be an inherited deficiency of glycogenolytic, glycolytic, or lysosomal enzymes. Quiroz-Rothe *et al.* (2002) recently observed the condition in the *longissimus* muscle of Anglo-Arab showjumpers and Andalusian dressage horses with back pain.

Tissue fat as an energy source

Horses described as 'moderately fat' require more energy for maintenance than do those in 'moderate condition'. The reserves of fat in the latter state are adequate as a source of energy for exercise, indicating that a relatively lean condition is satisfactory.

Exercise warm-up

Horses have a high aerobic capacity and rapid kinetics of gas exchange compared with other mammalian species that have been measured. The maximum rate of oxygen uptake per minute ($\dot{V}O_{2\max}$ ml/kg/min) in racehorses is double that of human athletes and $\dot{V}O_2$ increases very rapidly at the onset of high-intensity exercise. The kinetics of gas exchange are affected by a warm-up prior to high-intensity exercise which results in horses reaching steady state $\dot{V}O_2$ faster. Tyler *et al.* (1996) found that a warm-up for 5 min at 50% $\dot{V}O_{2\max}$ in Standardbred racehorses increased the aerobic contribution to total energy requirement from 72.4 to 79.3% when they were run to fatigue (1–2 min) at 115% of $\dot{V}O_{2\max}$. The maximal accumulated O_2 deficit was lower in the warm-up horse, i.e. 34.7 vs. 47.3 ml O_2 eq/kgBW. A warm-up is very desirable for maximum performance during high-intensity exercise, as it should allow greater use of aerobic energy sources and it may reduce the risk of injury. The effects are likely to be conferred by providing the time necessary for the redistribution of the blood supply and an increase in circulation rate.

Recovery

Recovery from intensive exercise is promoted by post-race aerobic trotting, compared with no exercise, as clearance of lactic acid is accelerated.

Bone remodelling and training

(See also 'Acid–base balance', this chapter.) The commencement of training causes a remodelling process to occur in the long bones to accommodate the stresses on the skeleton. This process involves the mobilization of bone salts and their redeposition. There is a risk of microfractures occurring if training is intensified too rapidly. Ca balance data indicate that bone density is low during the first two months of training and that Ca balance and bone density are still increasing after three to four months. When stalled four-month-old Quarter Horse foals were sprinted over only 82 m/day, five days a week for two months, there was an increase in the radiographic density of the third metacarpal (Hiney *et al.* 2002) (also see 'Ca and P in bone', Chapter 3). Diet must be adequate in Ca to allow for this physiological adaptation.

Causes of withdrawal from training

During the period 1990–1992 causes of illness or fatal injury among 496 racehorses in California were analysed (Johnson *et al.* 1994). Musculoskeletal injuries accounted for about 80% of TB and Quarter Horse submissions, among which forelimb fractures were prominent. Poor mineral nutrition is likely to have played some role in this statistic.

THE ENDOCRINE SYSTEM

An examination of Fig. 9.2 will reveal that the changing demands of the horse for energy are monitored by a number of endocrine secretions, or hormones. Where rapid changes are necessary, the signal for secretion by the appropriate glands is provided by the involuntary action of the autonomic nervous system reacting to environmental stimuli. This in turn brings about other essential changes in cardiac muscle and the smooth muscles of arteries, intestines etc. Endocrine secretions mediate their effects to a considerable extent by switching on and switching off some of the enzymes that regulate the chemical reactions in energy metabolism.

Insulin

The insulin molecule attaches to receptors on cell membranes to stimulate cellular uptake of glucose and glycogen synthesis and TAG 'clearance' (different cellular receptor), so that their blood concentrations are decreased after a meal. Insulin retards the breakdown of glucose. Horses accustomed to a high starch diet possess a high insulin sensitivity (so long as the β -cell function of the pancreas is normal) and therefore are more inclined to hypoglycaemic shock when subjected to a fast than are horses normally fed hay diets and accustomed to deriving blood glucose from other sources. The insulin sensitivity of a horse can be determined by measuring its glucose tolerance and insulin response to a given dose of starch or glucose. Low

sensitivity and poor tolerance increase the areas under the plasma response curve of insulin and of glucose. Large doses of glucose in insulin resistant, diabetic or fasted animals cause glycosuria.

In TBs, blood glucose normally peaks about two hours from the start of feeding. The peak value is 6.5–8.5 mmol/l (117–153 mg/dl) at 1.5–3 hours. There follows a gradual decline to a post-absorptive level of about 4.6–4.8 mmol/l (83–86 mg/dl). Ponies on roughage diets may maintain normal levels 1.4 mmol/l (25 mg/dl) lower than this. Blood insulin activity tends to fall slightly during work because of the catabolism of glucose, but some insulin is still probably required to ensure that glucose is available to the working muscle cell.

Glucagon

In order to sustain concentrations of blood glucose within normal limits, the influence of insulin is counteracted by that of glucagon. Whereas the former promotes uptake of glucose by all cells of the body (with the exception of brain cells), glucagon appears to focus its effects primarily on the liver and adipose tissue. It achieves an increase in blood glucose by stimulating those enzymes that cause a breakdown of liver glycogen (Fig. 9.2) and by encouraging gluconeogenesis. In this latter function it works in concert with other hormones discussed below, a particularly important task in roughage-fed animals. These other hormones are the glucocorticosteroids produced by the adrenal cortex and epinephrine and norepinephrine secreted by the adrenal medulla.

Adrenal hormones (catecholamines, glucocorticoids)

The rapid initiation of intense work necessitates an immediate response in terms of energy mobilization. This is brought about by sympathetic nervous activity, which not only causes splenic release of red cells but also stimulates the adrenal medulla to secrete epinephrine and norepinephrine (adrenaline and noradrenaline). The extent of this reaction depends on the intensity of the work load – that is, the faster the horse is running the greater is the secretion. The medullary hormones affect several tissues, increasing the mobilization of fatty acids from adipose tissue and stimulating the production of glucose with a rapid rise in the blood concentration by the breakdown of liver glycogen and by amino acid metabolism (Fig. 9.2).

The glucocorticoids secreted by the adrenal cortex are somewhat slower in responding to work demand and their secretion depends on a hormonal signal from the anterior pituitary. Moreover, they stimulate a rise in blood glucose and the accumulation of liver glycogen by promoting gluconeogenesis through the inhibition of protein synthesis. They also provoke the breakdown of depot fats to FFA and glycerol. Synthetic analogues of these secretions, when given repeatedly, have a comparable effect and cause muscular wasting. They also cause bone problems through an inhibition of Ca absorption from the gut. The stimulation from glucocorticoids of amino acid mobilization is expressed by excitation of transferase

enzymes, raising serum (Codazza *et al.* 1974; Sommer & Felbinger 1983; Essén-Gustavsson *et al.* 1984) and muscle (Guy & Snow 1977a) activities of ALT and AAT (see Fig. 9.2), observed after exercise.

In an analogous fashion to the response of glucagon, a reduction in blood glucose triggers the secretion of the glucocorticoids and their circulating level increases with agitation, trauma and psychological stress. Training leads to a greater adrenocorticoid response under such stress. This applies to sprint, endurance and other training so that normal concentrations of blood glucose are maintained more effectively in all circumstances. In contrast to the response of medullary hormones, amounts of plasma cortisol do not seem to be correlated with the intensity and speed of work. (NB: acute stress in the horse leads to an *increase* in circulating total cortisol, whereas chronic stress generally seems to decrease total plasma cortisol concentration. This may be due to the production of an adrenocorticotrophic-hormone (ACTH)-release inhibiting factor, which could be an endogenous opioid.)

THE VASCULAR AND RESPIRATORY SYSTEMS

Blood volume of horses is about 9.7% of body weight so that a horse weighing 560kg would contain around 51.2l (SG 1.06). Blood volume is important for both O₂ and heat transport so that a large plasma volume is accompanied by a considerable skin blood flow. This volume can increase by 30% over two weeks' training (Erickson *et al.* 1987). Other adaptations also increase blood supply to skeletal muscles. For example, resting glomerular filtration rate in TBs averages 3.3ml/kg/min, but, unlike the response in man, the value decreases by over 40% during exercise and the decrease is considerable even during a walk (Gleadhill *et al.* 2000). This decrease appears to be an adaptation to allow greater blood perfusion of muscle and skin.

The volume of blood discharged per beat (stroke volume) in a 560kg horse at rest would be 1.2l and as blood volume is proportional to body weight, cardiac output per ventricle ranges between 56 and 75ml/(kg × min) at rest. The need for such a flexible system can be appreciated when it is realized that O₂ consumption by skeletal muscle can increase 100-fold in strenuous exercise.

The blood of the horse is a fluid containing, by volume, 40–45% red cells (erythrocytes), (Arabians have a lower PCV than TBs, and racing dromedary camels a PCV < 30%), 1% white cells (leucocytes) plus platelets, and 54% plasma. The red cells have, as a major function, the transport of O₂ from the lungs to the muscles and other tissues. To accommodate an increased O₂ demand a reserve of red cells is held in the spleen. This splenic reserve is very large in TBs, so that they can increase the numbers of red cells in circulation by 30–60%. The adult TB can mobilize 6–12l red-blood-cell-rich blood into the central circulation, and cardiac output increases from 30l/min to amounts exceeding 300l/min at the onset of maximal exercise.

Thus, in heavy work, the O₂-carrying capacity of the blood may increase from rest by as much as 8.8 volumes %. Between rest and galloping at 700 m/min (26 mph), the O₂-carrying capacity changed from 15.9 to 21.4 volumes % in one study and from 16.35 to 25.19 volumes %, a 54% increase, in another study (Milne 1974) using TB, Quarter Horse, Arabian and Standardbred horses. The extent of change is increased by training and the effect of a change in capacity is augmented by a redistribution of the blood supply to skeletal and cardiac muscle and skin. The degree of splenic release tends to be proportional to speed; in one set of measurements the PCV rose 32% at 350 m/min (13 mph) and 55% at 700 m/min (26 mph) (Williamson 1974; for other observations see Figs 9.3 and 9.4). To assess the red-cell count and haemoglobin content of blood as affected by dietary inadequacies and other factors, the most consistent results are obtained after splenic release.

Blood and waste disposal

The supply of blood to muscles, increased during work, is the principal vehicle by which the waste products of energy metabolism, water, carbon dioxide and heat are removed from muscle cells. In the absence of their efficient disposal, pathological cellular changes would result. To facilitate this disposal, the capillaries of skeletal and cardiac muscle become dilated during increased work, while those in the visceral region contract. This in turn diminishes the digestion and absorption of nutrients and gives credibility to the theory that horses should not be fed before hard work (see 'Muscle energy reserves and feeding before exercise', this chapter).

Body temperature

At high work rates, heat production is 40–60 times basal levels, and body temperature can rise appreciably (Fig. 9.5). The mean muscle temperature of five Standardbred trotters increased from a normal of 37°C to 41.5°C during a race of 2100 m at a mean rate of 708 m/min or about 26 mph (Lindholm & Saltin 1974). Carlson (1983b) calculated that if a horse could work at a moderate intensity for one hour with an O₂ consumption of 30–40 l/min, the total waste heat would amount to 38 MJ. The principal mechanism for exhausting this waste heat from the body, when atmospheric humidity is not excessive, is the evaporation of sweat and of moisture from the surface of the lungs. The majority of heat is dissipated in this way through the skin, rather than the lungs, accompanied by electrolyte losses, accelerating the onset of fatigue. Dilatation of subcutaneous blood vessels diverts blood from skeletal muscle, contributing to a decrease in work capacity, at a cost influenced by the depth of subcutaneous fat (Webb *et al.* 1987b).

If circulatory adjustments fail to maintain heat balance, ventilation rate is increased (Fig. 9.6), which induces respiratory alkalosis. Training is important, as the rise in core temperature during exercise is proportional not to exercise intensity, but to the percentage $\dot{V}O_{2\max}$, which is increased by training, so ameliorating a temperature rise. Long low-intensity exercise on a treadmill at 30°C and 80% RH over 15

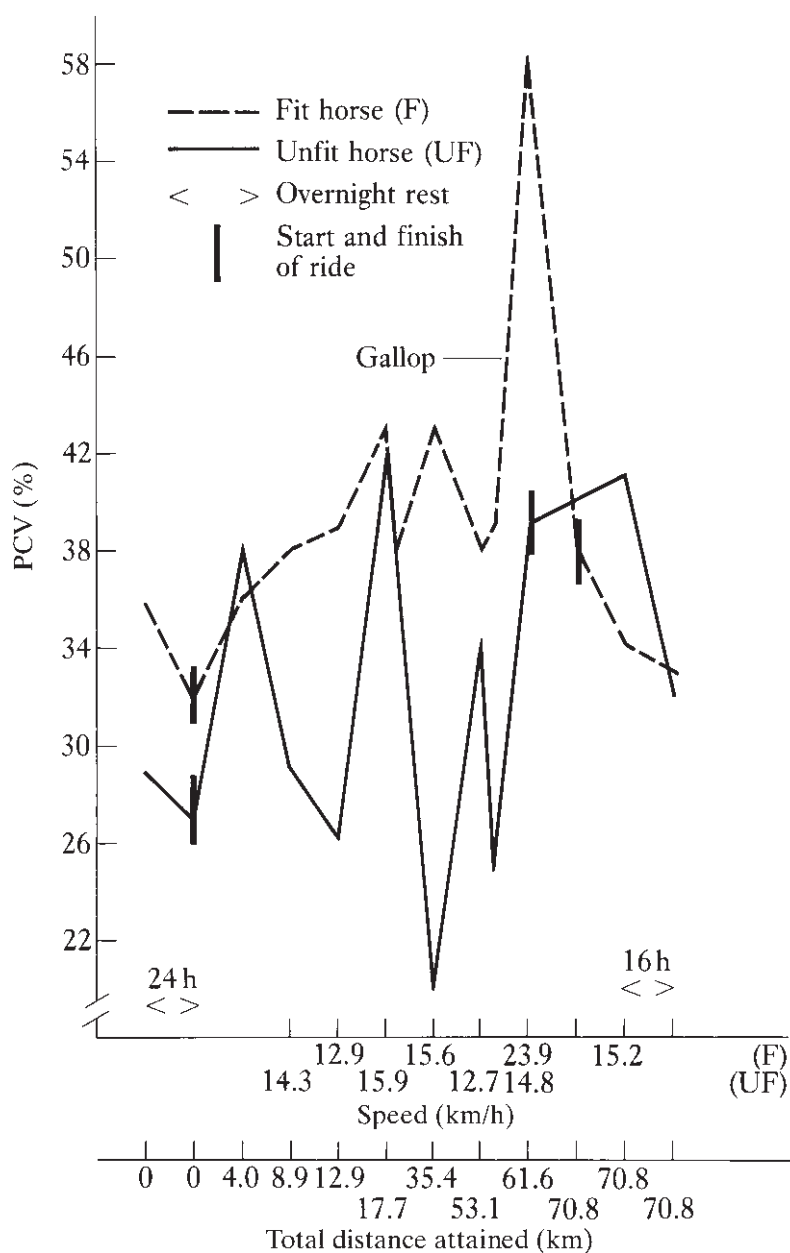


Fig. 9.3 PCV of the blood during an endurance ride by a fit Arab stallion and a less-fit pony gelding (Frape *et al.* 1979).

days gave horses an advantage in a three-day event at 30°C, 80% RH (Marlin *et al.* 1999). The advantage was assessed as a lower body temperature, lower metabolic heat production and decreased total fluid loss with sweating initiated at lower body temperatures.

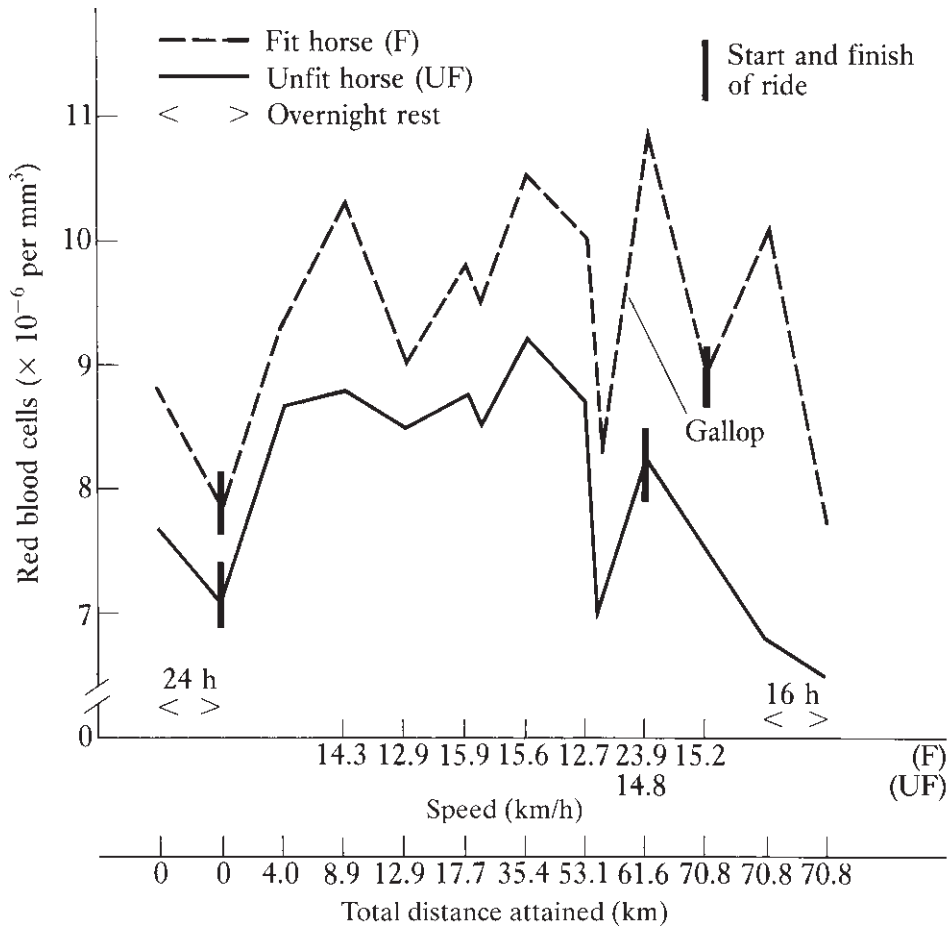


Fig. 9.4 Changes in erythrocyte count during an endurance ride in a fit Arab stallion and a less-fit pony gelding (Frape *et al.* 1979).

Following feed ingestion the amount of blood distributed to the GI tract increases, competing with the redistribution necessitated by a rise in heat production. Accommodation is achieved by augmenting cardiac output, redistributing regional blood flow, or by combining these mechanisms (Fig. 9.7).

Blood as a buffering system

Red blood cells contain haemoglobin, which not only carries O_2 but also acts as a buffer to the lactic acid produced in contracting muscles. This is probably why the pH of blood falls only during galloping, whereas the haemoglobin content rises appreciably during only moderate exercise. As haemoglobin is a major buffer, blood pH is changed very little for a considerable change in bicarbonate content. How-

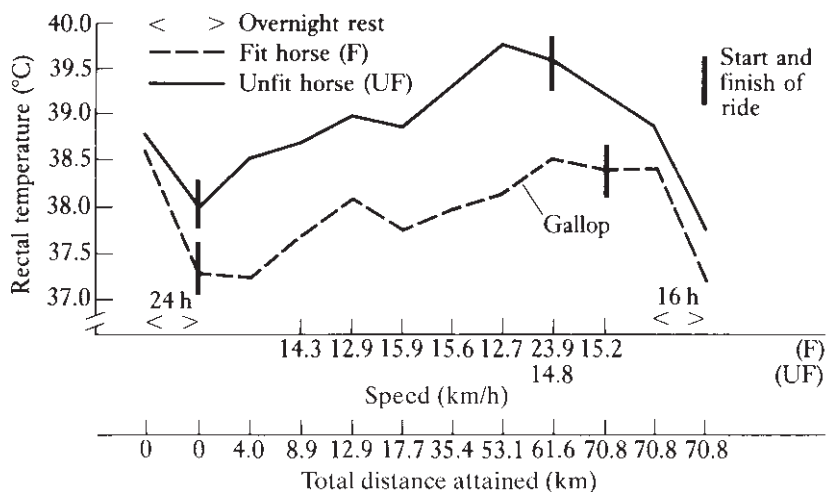


Fig. 9.5 Effect on body temperature of an endurance ride by a fit Arab stallion and a less-fit pony gelding in cool weather (Frape *et al.* 1979).

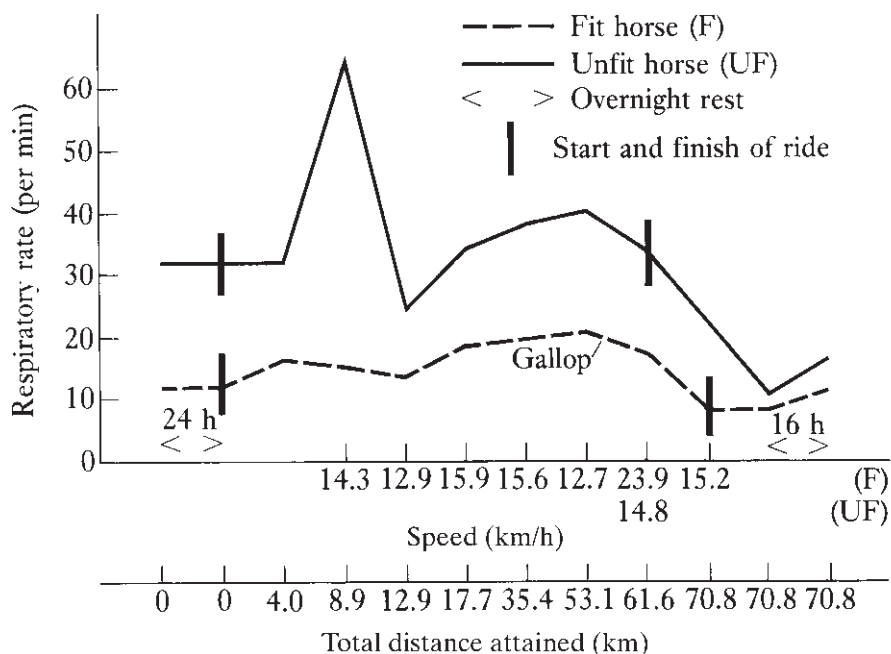


Fig. 9.6 Effect on respiration rate of an endurance ride by a fit Arab stallion and a less-fit pony gelding (Frape *et al.* 1979).

Feed consumption leads to:

- ↑ coeliac blood flow
- ↓ skeletal muscular blood flow, or
- ↑ cardiac work

Exercise leads to:

- ↑ skin blood flow
- ↑ water–electrolyte loss
- ↑ respiratory and heart rates
- ↑ pulmonary blood flow

Fig. 9.7 Heat flow and blood redistribution as a consequence of eating and exercise (Frape 1994) (↑, increase; ↓, decrease).

ever, in performing this function, haemoglobin carries less O₂. In extreme cases of acidosis the horse becomes cyanotic, or O₂-starved.

Blood glucose concentration

Blood glucose concentration is the expression of a dynamic balance of glycogen breakdown and glycogen synthesis and the production of glucose from other sources – amino acids, lactic acid and propionate (gluconeogenesis). The resting level is somewhat higher in horses intensely trained for sprint races. This state is brought about by the stimulation of the two systems leading to the formation of glucose and by increasing the efficiency of fatty-acid utilization, sparing glucose. Thus, blood glucose fluctuates throughout the day, rising in TBs from post-absorptive values of 4.7 mmol/l to a peak of approximately 6.5–7.5 mmol/l, two to three hours after a large feed of oats, following moderate work (those fed after strenuous exercise may show no plasma glucose response) (Frape 1989). In contrast, blood glucose concentrations reached values of between 4.7 and 6.4 mmol/l in ponies after receiving a pelleted meal to appetite, whereas after three hours of fast, the levels fell to between 2.8 and 5 mmol/l (Ralston *et al.* 1979). Plasma pyruvate also tends to increase with glucose, whereas fatty acids and glycerol are in lower concentrations.

The fluctuation between peak and resting levels of glucose varies with the type of diet, in that foods containing more grain and less roughage lead to higher peaks and lower troughs. Horses subjected to a rougher diet develop a greater faculty for gluconeogenesis and can therefore resist a depression in blood glucose more readily during a fast, but are unable to meet the demands of an excessive rate of exertion. Furthermore, breeds such as TBs, with higher insulin sensitivity, experience a greater fluctuation in blood glucose than do ponies given the same diet at similar times of the day.

Haematocrit, blood viscosity, blood volume and exercise-induced pulmonary haemorrhage (EIPH or 'bleeders')

Slight pulmonary bleeding seems to be a normal consequence of strenuous work and does not reflect any dietary abnormality, but simply reflects the physiological stress of a massive increase in nutrient and gaseous irrigation of muscle tissues. Pulmonary capillaries necessarily have very thin walls to allow rapid exchange of

respiratory gases across them and stress failure is greatly increased at high lung volumes. The capillary walls of TBs are not strong enough to withstand the stresses that develop as a result of the high capillary pressures accompanying extremely high cardiac outputs during strenuous exercise. There is some evidence that low environmental temperatures (range for most observations -10 to $+17^{\circ}\text{C}$) increase the risk (Lapointe *et al.* 1994).

In order for blood to travel freely through the capillary bed of muscles, it is essential that it retain its fluidity, and where the PCV exceeds 55% there is an exponential increase in blood viscosity. Providing horses with additional red blood cells before races can therefore be misguided. Optimization of blood flow to working muscles is dependent upon blood pressure, diameter and length of vessel, as well as blood viscosity. The high PCV of horses during exercise could be fatal in human athletes, but owing to blood's unique rheological properties that allow for optimal flow despite an elevated PCV during equine exertion, such a PCV is not fatal (McKeever 1998). However, changes in blood viscosity and other rheological factors may contribute to the rise in pulmonary artery pressure to which is attributed the onset of exercise-induced pulmonary haemorrhage (EIPH) (Weiss & Smith 1998).

Performance is impaired by the haemodynamic response to exercise in Standardbred trotters with red cell hypervolaemia (RCHV) (Funkquist *et al.* 2000). Exercise-induced pulmonary haemorrhage could be caused by the increase in red cell volume. This results in the high PCV and high total blood volume of RCHV horses that contribute to their high blood pressure in the pulmonary and systemic circulation during exercise (see Glossary for Hypervolaemia).

The importance of a low viscosity is recognized in horses subject to dehydration in hot climates and during long-distance rides (Figs 9.8 and 9.9). For this reason it may not be by chance that the PCV of Arab horses (and that of racing dromedary camels) (Fig. 9.3) is lower than that of other hot-blooded horses and that Arab horses are highly adapted to both hot climates and extended work. A voluminous splenic pool may therefore be a disadvantage for some purposes. On the other hand, there is evidence that a relatively greater increase in PCV and circulating red cell volume observed in a hot humid climate cf. a cooler one during racing confers a thermoregulatory advantage (Hargreaves *et al.* 1999) (NB: The critical feature is probably blood viscosity.)

Plasma albumin, a reserve protein and a major contributor to blood viscosity, displays a decreased concentration during training for reasons that may be understandable in this context. The dilutions achieved still provide adequate osmotic pressure and do not necessarily impute a dietary protein deficiency. In fact, the increase in dietary carbohydrate, normal at this time, may contribute to albumin catabolism.

Pulse, respiratory rate and fitness

Heart rate is linearly related to speed of the horse and varies between the approximate limits of 30 and 240 contractions/min. After a gallop at 700–800 m/min (26–

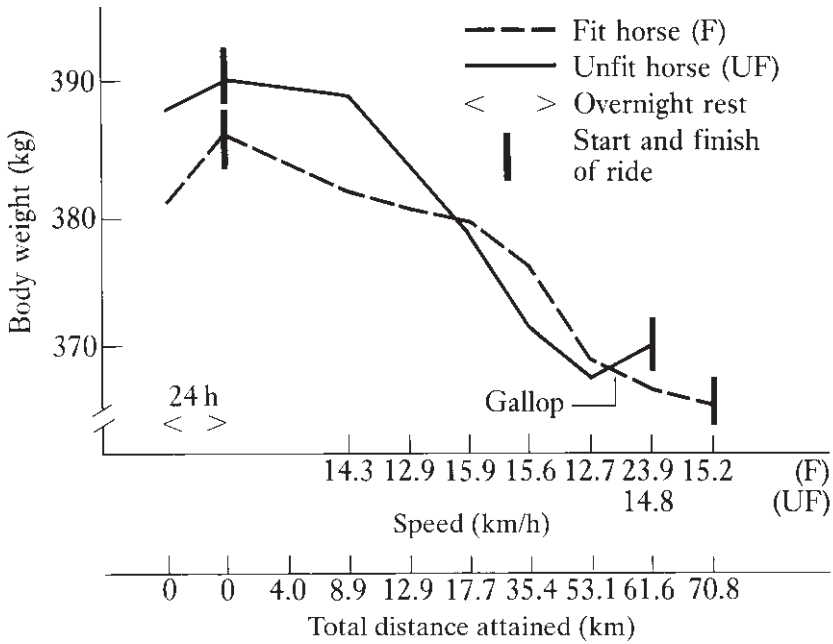


Fig. 9.8 Effect on body weight of an endurance ride by a fit Arab stallion and a less-fit pony gelding in cool weather (Frape *et al.* 1979).

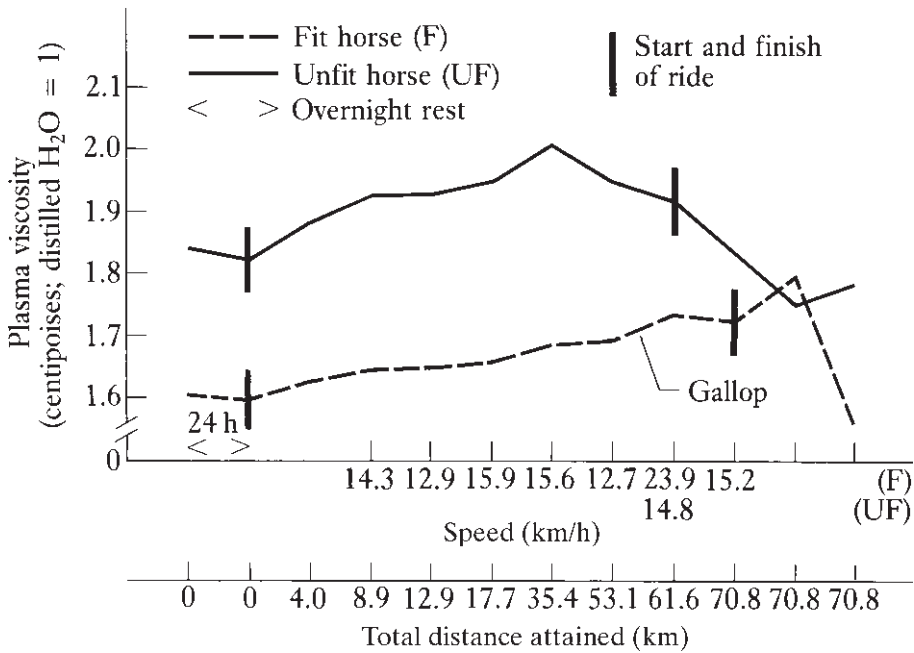


Fig. 9.9 Effect on blood plasma viscosity of breed and of an endurance ride by a fit Arab stallion and a less-fit pony gelding (Frape *et al.* 1979).

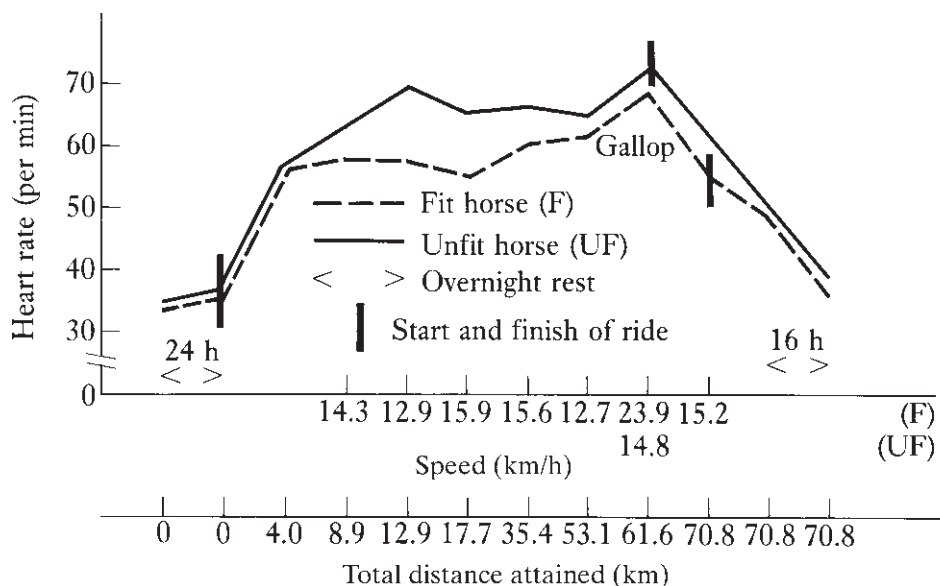


Fig. 9.10 Effect on heart rate of an endurance ride by a fit Arab stallion and a less-fit pony gelding (Frape *et al.* 1979). (NB: raised heart rate of unfit horse at 53–61 km may have resulted from anxiety when partner galloped off.)

30 mph) the rate may be as high as 240 beats/min with an output from each ventricle of 3–4 l/s. Heart rate, particularly after work, is a good indicator of fitness. In the pre-ride checks of endurance horses it is agreed that pulse rate should fall within 36–42 beats/min (Fig. 9.10) and respiration rate 8–14/min (Fig. 9.6). Both are higher in unconditioned horses. After an endurance ride and 20 minute rest the pulse rate should have fallen to less than 55 and the respiration rate to 20–25/min. In exhausted horses, the rate of both these is greater (tachycardia and hyperpnoea) and the occurrence of muscular spasms more likely.

The ratio of pulse to respiratory rate should fall within the limits of 2:1 to 5:1. During heavy exertion and heat stress the pulse and respiration rates have been known to rise to 85 and 170, respectively, a ratio of 1:2, that is, an inversion of the pulse:respiration rate ratio. Poorer horses and those suffering from adrenal exhaustion tend to exhibit lower heart:respiratory rates both before and after exercise. After a 20 minute rest during an endurance ride, horses exhibiting heart rates exceeding 70 or heart:respiratory rate ratios of less than 2:1 should be eliminated.

Hyperventilation may simply reflect a shortage of oxygen and normal respiratory acidosis (not commonly seen in endurance horses), or it may indicate an increase in body temperature (easily measured per rectum) brought on by hot weather or inadequate training, or both (Fig. 9.5). Alkalaemia normally follows a raised body temperature (see 'Body temperature', this chapter).

RESULTS OF EXERCISE

Sweat and dehydration

A horse working at a moderate intensity for one hour with an O_2 consumption of 30–40 l/min would need to dissipate 38 MJ waste heat (Carlson 1983b). The disposal of this quantity by evaporative processes alone would entail the loss of just over 15 l water. Although this is an oversimplification, it is a reasonable estimate of events in high environmental temperatures and low relative humidities. Sweat losses are quite modest in horses racing for distances of up to 3 km, but it seems that body-water losses in sweat (and urine), and from the lungs, during prolonged exercise can approach 10–12 l/hour. Of the total fluid losses, 20–30% is lost by expiration and the remainder is largely sweat. Hence a 500 kg horse could lose 30 l water in sweat, with a total loss of as much as 40 l during extended exercise.

Typically, body weight falls by 5–9%, principally from evaporative losses (Fig. 9.8), but the extent of loss depends on the level of fitness and the availability of water and electrolytes during the exercise. The evidence in Fig. 9.8 and Table 9.2 clearly indicates a greater rate of body-weight loss and of sweat production (carrying the fixed ions of Na, K, Cl, Ca, P and Mg) by an unfit horse than by a fit one, although in both horses the molar sum of cations approximately equals that of the anions in the sweat.

Water losses are more aptly compared with total body water, which, in a horse of 450–500 kg body weight, would amount to 8–14% of the total of about 300 l in extended exercise. Of this 300 l, about 200 l is in intracellular fluid (ICF) and 100 l is in extracellular fluid (ECF), made up of the water in blood plasma, interstitial fluid, lymph and contents of the GI tract.

Table 9.2 Electrolytes (mmol/l)¹ in evaporated sweat of horses participating in an endurance ride² and other data obtained from exercising horses.

Horse	Cl	Na	K	Ca	P	Mg	pH of venous blood
Fit at mid-ride ²	910	710	215	—	—	—	—
Fit at finish ²	1180	880	270	—	—	—	7.29
Unfit at finish ²	3060	2120	780	—	—	—	7.36
Harris ³	155	135	41	3	<0.3	2	—
Mean of other data ⁴	231	173	49	—	—	—	—
Hoyt <i>et al.</i> 1995 ¹	26	19	36	0.051	0.083	—	—

¹Hoyt *et al.* 1995a; data given as mmol/MJ DE consumed by miniature horses for work, above maintenance.

²Frape *et al.* 1979.

³Harris (1996), personal communication.

⁴Meyer *et al.* 1978; Carlson & Ocen 1979; Rose *et al.* 1980a; Snow *et al.* 1982.

Sweat electrolytes and their losses***Na, K and Cl***

The 100l of ECF contain 14 000–15 000 mmol readily exchangeable Na, representing nearly all the exchangeable Na of the body, the total Na content of which is about 1 kg. Of this total, 40% is located in the skeleton, and during extended Na depletion this depot appears to be partially mobilized. The 200l of ICF contain 20 000–30 000 mmol readily exchangeable K, most of the body's reserve. The bulk of the exchangeable Cl is present in the ECF, where it is the major fixed anion, but its concentration is substantially lower than that of Na – it is probably of the order of 10 000–12 000 mmol/100l.

Ca and Mg

Horse sweat is hypertonic with respect to plasma, containing 2–6 mmol/l Ca and 1–6 mmol/l Mg, indicating a loss of 2–7 g Ca and 0.6–4.0 g Mg in 30l. The sweat to plasma ratio ranges between 1:1 and 8:1 for Mg and 1:1 and 2:1 for Ca. However, Mg is mainly an intracellular cation and less than 1% is present in the extracellular fluid, so serum, or plasma, Mg is not a reliable indicator of body Mg status. Moreover, catecholamines are elevated during exercise. Raised blood adrenaline causes a net efflux of Mg from the intracellular space. Blood pH also influences the values of ionized Ca and Mg in plasma, as the extent of binding increases with increasing pH. Approximately 70–75% of plasma Mg is ultrafiltrable, of which the main portion (65% of total) is ionized and the remainder (8% of total) complexed with anions. Weiss (2000, personal communication) determined a decrease in total plasma concentrations of Ca and Mg following a trot. This decrease reflected the sweat loss, as urinary Mg is not increased in exercising horses (Meyer *et al.* 1991). Body stores of Ca and Mg are generally sufficient to replace sweat losses, but serum concentrations of Ca and Mg also reflect dietary supply and a liberal supply may prevent critical adverse clinical consequences of exercise (see also 'Hypo- and hypercalcaemia and stress tetany', Chapter 11).

Sweat losses during work

The primary route of fixed-ion loss by working horses is sweat produced for the purpose of preventing an excessive rise in body temperature, so that dehydration through sweating entails a loss of both water and electrolytes with a contraction of the volume of body fluid. Hoyt *et al.* (1995a) found relatively greater losses of K than of Na and concluded that dietary needs during exercise were increased over the basal dietary supply by threefold, sevenfold and sixfold, respectively for Na, K and Cl. It would be important to establish the validity, or reason, for the differences in relative loss of Na and K.

Changes in the composition of the blood plasma depend on the proportions lost of each of these constituents and of water and on the movement of ions into and out

of ICF space. Extended exercise, with minimal water consumption, leading to dehydration, will normally precipitate a substantial reduction in the concentration of plasma Cl. Little change is frequently detected in the amounts of K and Na, although hypokalaemia is not rare. The explanation for the hypochloraemia is revealed by a comparison of the Na and Cl contents of sweat (Table 9.2) with their respective concentrations in blood serum (Table 3.1), demonstrating that a much greater proportion of Cl than of Na in body fluid is lost. Extended work, in hot dry weather, by a 450–500 kg horse expressing the above plasma changes may yield losses of as much as 35 l of water, 3500 mmol Na, 1500 mmol K and 4200 mmol Cl (equivalent to 80 g Na, 59 g K and 149 g Cl). The loss of Na, for example, represents over 200 g of sodium chloride, that is much more than a horse would eat in a day. These values are at variance with the relatively higher values for K reported by Hoyt *et al.* (1995a), which are equivalent to 0.43 g Na, 0.93 g Cl, 1.41 g K, 2.03 mg Ca and 2.56 mg P/MJ DE consumed for work above maintenance.

The total concentration of electrolytes in sweat is higher than that in blood plasma, so a decline in the plasma concentration, despite considerable dehydration, is readily understandable. Most studies have revealed a decline in plasma electrolytes during endurance rides taking place in hot weather. For example, Carlson *et al.* (1976) reported reductions of 4.2 mmol Na/l, 0.9 mmol K/l, 10.3 mmol Cl/l and no change in Ca. On the other hand, the changes can be variable depending on such factors as relative losses of water to electrolytes, plasma pH changes and time of collection after the ride. In horses losing a considerable amount of water, Rose *et al.* (1977) detected increases of 6 mmol Na/l and 0.3 mmol K/l plasma, no change in Ca and a decrease of 6.8 mmol Cl/l.

Glycogen stores are depleted after extended exercise. The administration during recovery of glucose solutions by nasogastric tube has not stimulated glycogen resynthesis, whereas a NaCl solution of 4.5 g/l or 9 g/l has (Vervuert *et al.* 1999). Moreover, NaCl was more effective in restoring plasma volume and normochloraemia (Coenen *et al.* 1999). Nevertheless, the rise in plasma Na⁺ may increase blood pressure and secretion of natriuretic peptide. During a second exercise test, two hours following the recovery period there was a decrease in blood glucose and higher blood lactate in horses that had been treated with glucose, whereas, in those that had been treated with electrolytes, blood glucose remained unchanged and there was a pronounced FFA response (Vervuert *et al.* 1999).

Thirst, drinking and osmotic pressure of blood

Thirst is in part controlled by the osmotic pressure of the blood and therefore it is frequently necessary to rectify electrolyte loss (Tables 9.3 and 9.4) before dehydrated horses will drink adequate quantities of water. Electrolyte mixtures are widely available commercially. If the horse is at the same time acidotic, then plasma K may rise, despite a K deficit, as intracellular K is exchanged for H⁺ ions. Subsequently, renal losses of K may increase, ultimately causing severe K depletion. A

Table 9.3 Composition (mmol/l) of various electrolyte solutions for i.v. use¹ (Rose 1981).

	Na	K	Ca	Mg	Cl	Glucose	Bicarbonate	Lactate	Acetate	Gluconate	Propionate
0.9% NaCl	154	—	—	—	154	—	—	—	—	—	—
5% Dextrose	—	—	—	—	—	278	—	—	—	—	—
Hartmann's solution	131	5	2	—	112	—	—	28	—	—	—
Ringer's lactate	130	4	3	—	109	—	—	28	—	—	—
Normosol R ²	140	5	—	1.5	98	—	—	—	27	23	—
Dilusol R ³	140	5	—	1.5	98	—	—	—	27	23	—
Normosol M ²	40	13	—	3	40	278	—	—	16	—	—
Balanced electrolyte solution ⁴	137	5	3	3	95	—	—	—	27	—	23
Solution to treat acidosis ⁵	137	20	—	—	97	—	60	—	—	—	—
5% NaHCO ₃ (hypertonic)	600	—	—	—	—	—	600	—	—	—	—
5% Dextrose saline (hypertonic)	154	—	—	—	154	278	—	—	—	—	—

NB: Intravenous solutions should contain a bicarbonate precursor (lactate, acetate, gluconate or propionate), as the sole use of fixed bases such as chloride can cause metabolic acidosis, hypokalaemia and hyperchloraemia.

¹ Give only 1–2 l/h of solutions containing 5% dextrose, but up to 3–5 l/h for other solutions, all of which should be at 37°C.

² Abbot Laboratories, Illinois, USA.

³ Diamond Laboratories, California, USA.

⁴ Merritt (1975) in Rose (1981).

⁵ Rose (1979) in Rose (1981).

Table 9.4 Composition (g/kg DM) of mixtures for administration by stomach tube and as a daily supplement.

Mix*	Glycine	Sodium chloride	Monopotassium phosphate	Magnesium sulphate	Potassium chloride	Calcium carbonate	Calcium gluconate	Sucrose or glucose*
(1)	470	270	190	13			57	
(2)		325			325	175		175
(3)		170			70			100

NB: Mixes (1) and (2) may be squirted as a thick slurry to the back of the mouth *after* water has been given, to rectify a water deficit and thirst. Mix (3) is a suggested simple mixture to be given in the amounts shown daily as a dry supplement mixed with bran or other palatable dry material to a 500 kg horse during periods of extreme effort in hot weather. Fresh water must be freely available. (NB: It is usually more 'palatable' dry than as a solution.)

In order to provide approximately isotonic solutions, add 230 g mix (1) to 6 l water, 120 g mix (2) to 6 l water given every 2–3 h for a 500 kg horse.

*May be replaced by molasses.

further discussion of the means of assessing K status, and the major causes of depletion and their treatment are given in Chapter 11.

Horses should be allowed to drink frequently during extended work and at least two minutes should be allowed on each occasion. If the weather is very hot, then a drink at least every two hours is desirable. Hard work diverts much of the blood supply to the skeletal muscles away from the splanchnic bed of blood vessels serving the GI tract. It is thought that this inhibits the efficient absorption of water, so that the amounts consumed on any one occasion should be relatively small. The consumption of large volumes is also to be avoided because of the large difference between it and blood in osmotic pressure. After work, very dehydrated animals should be given about 4.5 l (1 gallon) every 15 min, preferably containing 30 g electrolytes. If the horse will not drink, administration by stomach tube is sometimes necessary. Dehydration is frequently accompanied by coldness and fatigue, muscular tremors, colic, thumps, lack of appetite and a low pulse:respiration-rate ratio. Severely dehydrated animals are sometimes given a 5% glucose–electrolyte solution i.v., while heart rate is monitored, when their ability to absorb fluid from the gut is in doubt (Table 9.3).

Where there has been a contraction of blood volume, the administration of electrolytes has only a transient effect on increasing that volume. Nevertheless, rectification of losses and of the acid–base balance must be considered. The total osmotic pressure of the blood depends to a large extent on the colloids it contains, the principal ones being proteins, more especially albumin. However, protein loss during work will be minimal and reflect only its metabolism as an energy source apart from very slight losses through pulmonary haemorrhages. The rebuilding of energy reserves, particularly in respect of muscle glycogen, will take several days after extended hard work.

Fatigue and interval training

Fatigue during extended exercise partly results from a decline in blood glucose concentration. The rate of this decline is affected by velocity, by the extent of glycogen stores, by dietary manipulation that spares glycogen mobilization and by training that promotes a greater use of both fat and glycogen during maximal exercise (Snow & Mackenzie 1977a). Fatigue is first exhibited by a decrease in speed during the course of running. Contributory factors to fatigue therefore also include a loss of muscle ATP, depletion of muscle glycogen, metabolic acidosis and blood lactate and ammonia accumulation. Fatigued horses may still possess a mean of 400–500 mmol glycogen/kg muscle tissue, but the muscle fibres used predominately in submaximal extended exercise, that is, the type I fibres (slow-twitch oxidative fibre) may be almost depleted and the type IIa fibres (fast-twitch oxidative) show a reduction in glycogen content.

To obtain a large reserve of glycogen, there is a maximum advisable for sprint training. Excessive training leads to an increase in the time over 1200 m. This is associated post-exercise with an increase in blood lactate, a decrease in plasma cortisol concentration and packed cell volume and decrease in body weight and $\dot{V}_{200}$ (Hamlin *et al.* 2002). Tapered training over seven days before a 2400 m individual timed trail reduced fatigue and improved performance in comparison with constant training. The introduction of interval training to the tapered system increased the risk of injury (Shearman *et al.* 2002). Although glycogen loss is pronounced in sprint exercise and fat is a major energy substrate in submaximal extended exercise, glycogen loss inevitably occurs during extended exercise.

The object of interval training is to increase the volume of work accomplished in a single training session by providing exercise in bouts separated by recovery periods. This system allows some recovery of the muscle glycogen reserves and a reduction in heart rate and plasma lactate concentration during rest periods. The reduction in plasma lactate will be accelerated by trotting the horse between bouts, rather than by walking or cantering. Repeated high-intensity interval training has caused loss of performance capability, body condition and weight and so should be carefully monitored.

Measurement of fitness and exhaustion

A medium-paced canter is normally accomplished with aerobic respiration so that the ratio of lactate to pyruvate in the blood is unchanged. However, during galloping, the ratio rises sharply, and an effect of training is to induce smaller changes in blood lactate and in the lactate to pyruvate ratio. Blood lactate concentration may nevertheless be unrelated to racing speed, but as it is a relatively strong acid it tends to be correlated with blood pH. Blood lactate concentration is partly an expression of adaptation to training, Persson (1983) proposes that the estimation of blood lactate at a work-induced heart rate of 200 beats/min could be used to gauge fitness in training, where lower lactate concentrations down to 2 mmol/l would reflect

Table 9.5 Effects of racing 1900–2500 m on concentrations of blood lactate and acidity and their relationship to exhaustion (Krzywanek 1974).

	Blood lactate (mEq/l)			Blood pH		
	Before race	After race		Before race	After race	
		Immediately post	15 min post		Immediately post	15 min post
Exhausted	0.58	23.14	24.37	7.379	7.086	7.105
Not exhausted	0.63	17.99	16.92	7.379	7.164	7.213

greater fitness. Heart rate would be measured by telemetry on the track and the results would be unaffected by track conditions that affect speed.

In addition to dehydration and electrolyte depletion, blood acidity is a dominating factor in determining exhaustion (Table 9.5) and serum levels of the enzyme CK provide an indication of the severity of exercise and metabolic acidosis. In one study, where horses raced at the rate of 700 m/min (26 mph) serum AAT rose 50%, serum Ca rose 13%, but serum CK rose 227% (Williamson 1974). It is thought that the increase in serum concentration of muscle enzymes after exercise is explained by an increase in the permeability of muscle cell membranes, owing to hypoxia. Thus, inadequate training, or severe work loads, will induce a greater increase in the serum concentration of CK.

Ammonia and the alanine vehicle

The accumulation of ammonia (NH_3) in the blood of exercised horses probably contributes to fatigue, although the experimental evidence is weak. Ammonia causes pyruvate to accumulate, which in O_2 -limited states, is converted to lactate, resulting in a decrease in muscle pH. Blood ammonia concentrations have been decreased by dietary monosodium glutamate (MSG) supplementation. Plasma ammonia and uric acid concentrations rise and attain maxima during recovery (Harris *et al.* 1987; Miller-Graber *et al.* 1987; Miller-Graber & Lawrence 1988), signifying an increased rate of AN cycling. A greater demand for ATP is partly met by the myokinase reaction ($2\text{ADP} \rightarrow 1\text{ATP} + 1\text{AMP}$) (Fig. 9.11; Frape 1994). Deamination of AMP, which supplementary NaHCO_3 may ameliorate, produces NH_3 and IMP, yielding uric acid (Harris *et al.* 1987) and alanine as the ammonia vehicle. Additional dietary protein may, or may not, aggravate this (Fig. 9.11).

Summary of fatigue

The first requirement of exhausted dehydrated horses is water, followed closely by electrolytes. Bodily energy resources must then be rejuvenated, and, if the weather

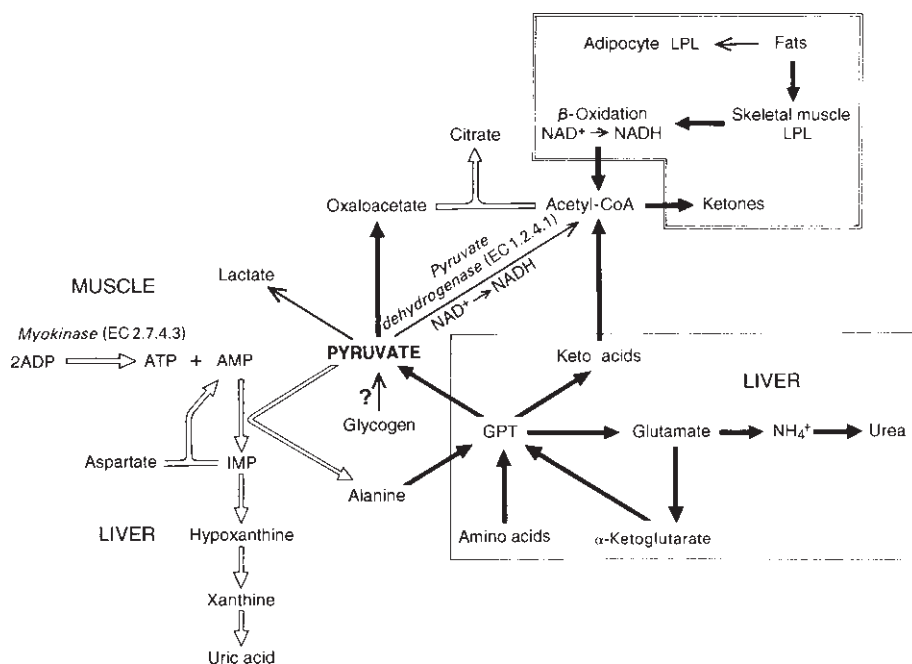


Fig. 9.11 Key: Proposal for reactions stimulated (➔), not measurably influenced (⬚) or suppressed (➞) by high protein or high fat diets during equine exercise of moderate intensity in the post-absorptive state. Reactions proposed to be stimulated by dietary fat (▣) and by dietary protein (▢). GPT, glutamic-pyruvic-transaminase (EC 2.6.1.2); LPL, lipoprotein lipase (EC 3.1.1.34); IMP, inosine monophosphate. (Frape 1994.)

is cold, the horse must be kept warm but not hot. Metabolically some ATP may be reformed by the myokinase reaction in the presence of pyruvate, but generally the fuels for ATP synthesis are depleted. After normal hard work a horse should be cooled by gentle exercise of the muscles through walking, ridding the muscles of waste products, but access to light grazing or hay should not be ruled out. After this relaxation of 1–1.5 hours, tepid water should be given before a light meal of concentrates. It can be concluded that exhaustion during long-distance work is an expression of nutrient depletion whereas during sprint work it is principally the result of raised blood lactic acid and a consequential fall in blood pH.

BLOOD ACID-BASE BALANCE

What is an acid?

An acid is a substance, such as lactic acid, that yields hydrogen ions in solution. The acidity of blood or other solutions is expressed as the pH (the negative logarithm of the H⁺ ion concentration). Acids and bases are produced during the metabolism of nutrients, and abnormalities in the acid-base balance result from dysfunction, or

overloading, of general metabolism and respiration. The normal pH of arterial blood is 7.5 and that of venous blood 7.4. The blood carries CO_2 to the lungs, partly in the form of weak carbonic acid (H_2CO_3), one of the principal acids of fizzy drinks. It and haemoglobin act as the principal buffers in blood; that is, they prevent the pH from shifting appreciably and so prevent death from this cause. In the plasma, CO_2 reluctantly and slowly forms H_2CO_3 , but on diffusing into the red cells this reaction is accelerated 13 000-fold by the presence there of the enzyme carbonic anhydrase. Despite the accelerated change, only 1 part in 800 of CO_2 forms H_2CO_3 . This is described in Fig. 9.12, which shows that nearly all this H_2CO_3 dissociates into H^+ and bicarbonate (HCO_3^-) ions. The former are partly buffered by haemoglobin and the latter to a large extent diffuse back into the plasma so that around 20 times as much CO_2 is carried as HCO_3^- as remains in the form of dissolved gas. Now, the dissociated form of H_2CO_3 in blood solution is in a constant (K) proportion to the undissociated form, as shown below:

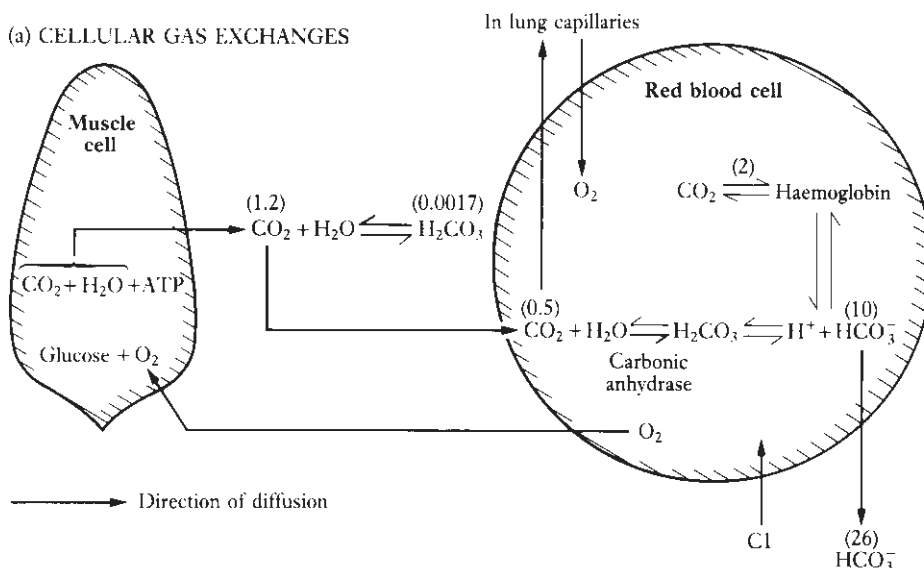
$$\frac{[\text{H}^+] \times [\text{HCO}_3^-]}{\text{H}_2\text{CO}_3} = K$$

If acid is produced during muscular activity, or during intestinal colic (see Chapter 11), H^+ ions in the numerator increase and these react with HCO_3^- forming CO_2 and water (Fig. 9.12). In this process the HCO_3^- concentration falls, but the H^+ ion concentration does not rise as much as it would in the absence of the HCO_3^- buffer, and so the ratio $\text{HCO}_3^-:\text{CO}_2$ governs the pH of the blood.

Acidosis and alkalosis

Fatigue during exercise is associated with a deviation of blood pH from the ideal range, causing metabolic acidosis or, with overheating, respiratory alkalosis. In lactic acidosis there is increased production of H^+ ions owing to an O_2 debt. With overheating there is increased loss of CO_2 , resulting from a high respiratory minute volume (Frape 1994). Metabolism for all important functions occurs most efficiently when arterial pH approaches the normal value of 7.5. Deviations are associated with losses of important ions through the kidneys and intestinal tract, causing a burden on metabolism which has eventually to be rectified. A lowering of pH results from an excessive rate of acid production during exceptional work and from disease states of the intestinal tract and associated organs, kidneys and lungs in particular. Organic acids produced in metabolism have only short-term consequences as they should be ultimately disposed of by metabolism and respiratory compensation. Fixed acids and bases (those that cannot be metabolized to CO_2 and water and exhaled) absorbed from digesta have a longer term influence and attention has been focused on the fixed cations and anions present in diet and their deducible effects on acid–base balance. Bone acts as a buffer to prolonged dietary imbalances of this kind. An acid diet, containing an excess of fixed anions, leads to bone resorption and osteoporosis so that renal excretion of those anions may proceed.

(a) CELLULAR GAS EXCHANGES

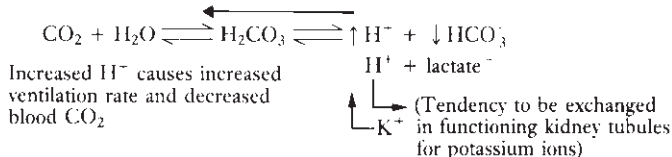


Note:

- Numerical values are approximate concentrations (mmol/l) of various forms of CO_2 .
- Total concentrations of CO_2 $[\text{CO}_2] = \text{dissolved } [\text{CO}_2] + [\text{HCO}_3^-] + \text{protein-bound } (\text{CO}_2)$.
- The enzyme carbonic anhydrase has a vital influence on CO_2 -carrying capacity of blood, which is therefore a function of the number of red cells.

(b) PLASMA REACTIONS

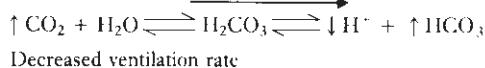
(1) Metabolic acidosis

Note: O_2 debt

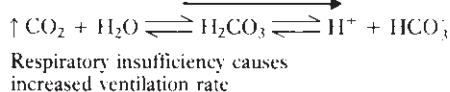
(2) Metabolic alkalosis

Note: HCO_3^- generated replacing Cl^- lost in sweat

K^+ lost in sweat is replaced in renal tubular filtrate by H^+

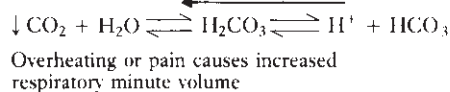


(3) Respiratory acidosis

Note: Plasma $[\text{H}^+]$ tends to rise

(4) Respiratory alkalosis

Note: A decrease in $[\text{H}^+]$ and possibly $[\text{HCO}_3^-]$ results from respiratory changes



↔ Increased direction of reaction

↑ ↓ Initial changes in plasma concentration of metabolite

Fig. 9.12 Effects of metabolic state on carbonic acid in blood plasma and associated relationships in muscle cells and red blood cells.

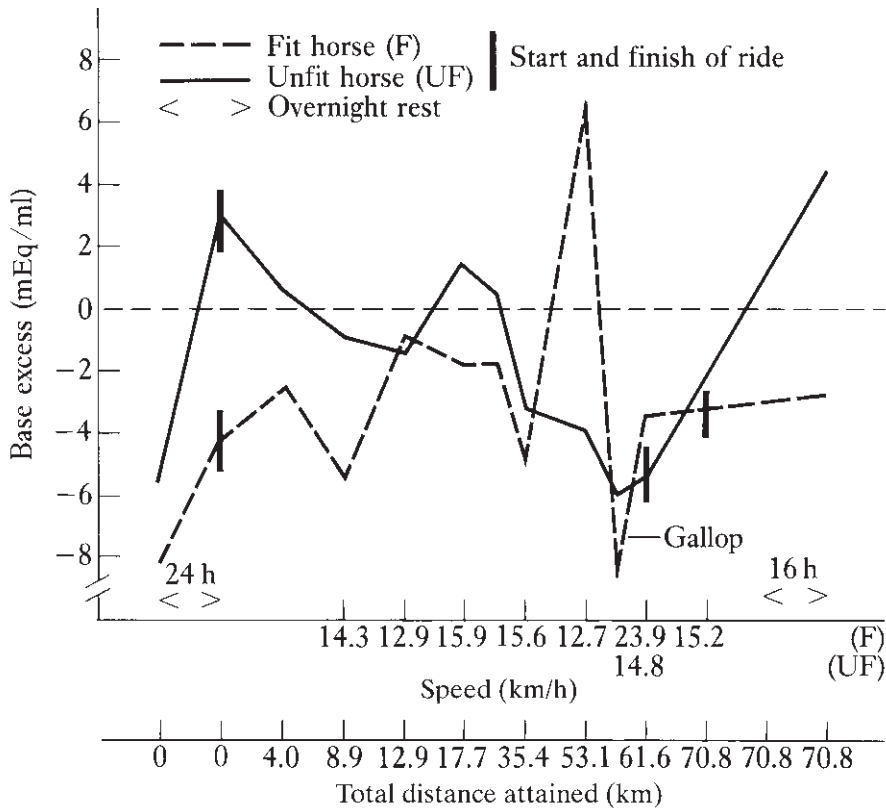


Fig. 9.13 Changes in plasma base excess (BE) during an endurance ride by a fit Arab stallion and a less-fit pony gelding (Frape *et al.* 1979). BE of the unfit horse declined from the start. Its respiration rate was consistently the higher, which could imply that a higher ventilation rate was removing more CO_2 from blood and carbonic acid reaction was moving to left (Fig. 9.12b '(4) Respiratory alkalosis' equation). Lap eight was run at lowest speed (Fig. 9.10) and, although respiration rate of the unfit horse did not fall (Fig. 9.6), the low rate of the other horse immediately allowed the BE to rise. This lap was followed by a rest period before the fit horse was galloped.

Base excess

A quantitative measure of the acid–base status of a horse, as affected by metabolism, health and diet, is known as the base excess (BE) (Fig. 9.13). This is the base content of the venous blood measured by titration with a strong acid to a pH of 7.4 at normal CO_2 tension. Base deficit is the same as negative BE and its measurement requires titration with a strong base, again to a pH of 7.4. At that pH, BE is zero and plasma bicarbonate equals approximately 22–25 mEq/l. In the normal horse, bicarbonate should be in the range of 24–27 mEq/l and the BE 2–5 mEq/l.

Function of the lungs and kidneys

The lungs have a vital short-term role in the acid–base balance by providing a route for the excretion of carbonic acid (Fig. 9.12). Although the kidneys also function in

this role, quantitatively they are insignificant, as the lungs dispose of 200 times more of the acid. However, the kidneys fulfil a long-term role in disposing of nonvolatile acids and bases from the diet [see 'Dietary base excess (BE) and "fixed" cation-anion balance (DCAB)', this chapter]. In this balance, and in that of acid-base, an important principle is disclosed. This is the principle of *electroneutrality*, in which all urinary and lung excretion and in all movement across normal cell membranes in the horse no electrical charge can accumulate, as in a battery. The number of anions has to correspond with the number of cations moving in the same direction. This may lead to pH changes of various bodily fluids and the concept forms a basis for an understanding of the issues involved in acid:base and cation:anion balance.

Like most laws, this one is only approximately true as it may conflict with that of iso-osmolarity (see Glossary), in which all bodily-fluid compartments (among which water is exchangeable through semipermeable membranes) approach isotonicity. In achieving this, a small voltage gradient occurs as, for example, between intracellular fluid in the muscles and extracellular fluid. These principles play a central part in muscular ailments, such as 'tying-up' (see Chapter 11). In so far as their acid-base status is concerned, horses are subject to four types of abnormal metabolism. Only those conditions relating to physical work will be discussed here.

Metabolic acidosis

In strenuous exercise, an O₂ debt, with a tissue accumulation of lactic acid, occurs to varying degrees. It is also possible that this form of acidosis may exist in combination with a B-vitamin deficiency, causing an incomplete metabolism of pyruvic acid, but this proposition has not yet been substantiated. The build-up of H⁺ ions causes hyperventilation and thus an increase in respiratory minute volume with a fall in blood partial pressure of carbon dioxide (PCO₂). This shifts the carbonic acid equation to the left (Fig. 9.12), which brings about some respiratory compensation of the acidosis and prevents an excessive fall in pH. In an analogous situation, the raised H⁺ ion concentration and pain of laminitis will have a similar respiratory effect so that in its advanced state respiratory alkalosis supervenes. Sodium bicarbonate, at a rate of 250–300 g (3000–3600 mEq) over 24 hours is sometimes given as a therapy for metabolic acidosis, but its unconsidered use can produce untoward effects. Moreover, the feeding of excessive sodium chloride (NaCl) will exacerbate an exertion-induced acidosis (Hinchcliff *et al.* 1993).

Metabolic alkalosis

Alkalosis can occur in chronic laminitis as well as from exhaustion after long-distance cross-country work in hot weather. A reduction in ventilation rate in the worst-affected horses may, however, induce slight acidosis. During extended work a depletion of body potassium and chloride develops in varying degrees. H⁺ ions are excreted as a substitute for the depleted potassium, and bicarbonate fills the anion

gap after loss of chloride. Tetanic spasms may sometimes result *in extremis* from a decreased availability of ionized calcium brought on by alkalosis. Unfit horses may present signs of adrenal exhaustion. The consequent failure to secrete aldosterone precipitates an excessive urinary loss of sodium with potassium retention and a further decline in well-being. Sodium bicarbonate therapy would severely aggravate alkalosis, and the provision of a balanced electrolyte and glucose solution containing sodium, potassium and chloride (see also Chapter 11) is to be recommended. The want of potassium is not truly attested by its plasma level, as a consequence of a shift from intracellular to extracellular space (see page 458). Electrolyte losses and hypocalcaemia, related to a raised pH, in exhausted endurance horses, are associated with a condition known as synchronous diaphragmatic flutter, in which heart and respiratory contractions coincide. In addition to electrolytes, calcium gluconate solutions are frequently given i.v. (Table 9.3).

Respiratory acidosis

This is the build-up of blood CO_2 (hypercapnia) which arises with the respiratory insufficiency of sprint work. It is obvious that the condition may also obtain with several diseased states of the lungs, including respiratory allergy, and the resulting decline in oxygen tension (hypoxia) in the blood may cause metabolic acidosis.

Respiratory alkalosis

Hard work in hot weather brings about overheating which directly affects the respiratory centre of the brain, causing rapid breathing. Rapid ventilation rate flushes CO_2 from the blood. The pain of colic and laminitis also accelerates respiration rate with similar consequences. The effect is to induce a slight rise of blood plasma pH (Fig. 9.14), although an absence of change in endurance horses may reflect a concomitant fall in both CO_2 and HCO_3^- .

Factors controlling plasma pH

Plasma $[\text{H}^+]$ is regulated by several independent variables, especially:

- partial pressure of CO_2 , i.e. PCO_2 , controlling $[\text{H}^+]$ and $[\text{HCO}_3^-]$;
- strong ion difference (SID), i.e. $[\text{Na}^+] + [\text{K}^+] - [\text{Cl}^-] - [\text{lactate}^-]$; and
- weak electrolytes $[\text{A}_{\text{total}}]$, represented by [albumin].

A decrease in plasma SID is normally associated with a decrease in plasma pH. This points to an increase in plasma $[\text{H}^+]$, implying there is insufficient $[\text{HCO}_3^-]$ available with which it can combine to maintain the constant K in the bicarbonate equation (see ‘What is an acid?’, this chapter). (NB: The function of dosing with NaHCO_3 is to provide that bicarbonate ion. Also, the Na^+ ion balances the lactate $^-$ ion, the concentration of which in plasma does not necessarily decline.)

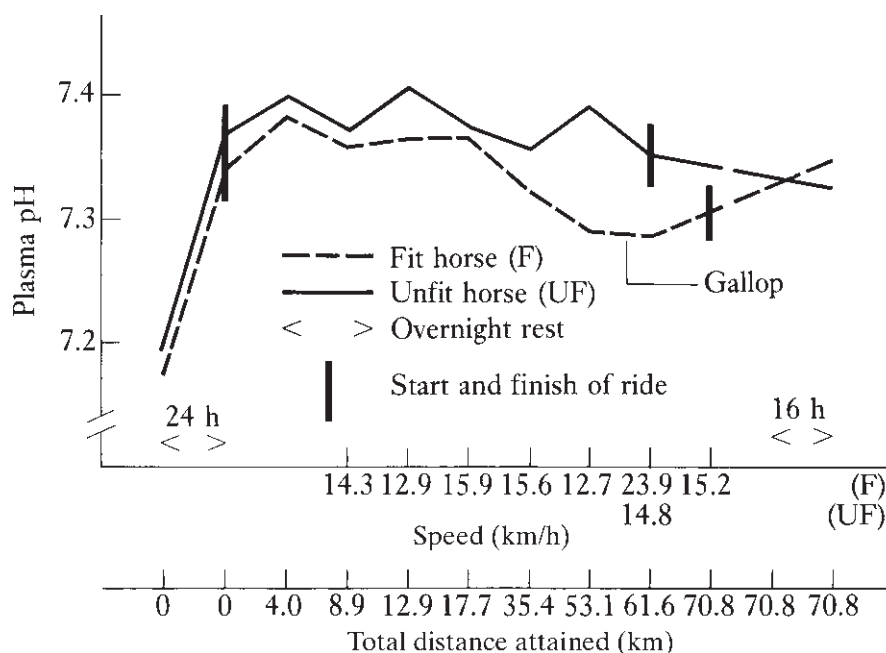


Fig. 9.14 Effects of an endurance ride on venous blood plasma pH in a fit Arab stallion and a less-fit pony gelding (Frape *et al.* 1979).

DIETARY BASE EXCESS (BE) AND 'FIXED' DIETARY CATION-ANION BALANCE (DCAB)

Dietary BE

Mineral nutrition has a role in acid-base balance. This follows from the premise that excess base, or acid, in feed may be estimated as the difference between the sums of mineral cations and anions, approximated by:

$$\text{Dietary BE (mEq/g)} = (\text{Na} + \text{K} + \text{Ca} + \text{Mg}) - (\text{Cl} + \text{P} + \text{S})$$

The effect that diet and metabolic acid production have on plasma BE is shown in Appendix D. Typical horse diets have a dietary BE of 200–300 mEq/kg estimated from the fixed-ion content. Thus, for a horse consuming 10 kg feed/day, the BE would amount to approximately 2500 mEq, which is similar to that provided by 200 g sodium bicarbonate – a quantity frequently given in therapy over a period of 24 hours. Horses with a serious deficit of bases may require, over that period, double the amount contained in a normal ration. The dietary optima assume that the protein content is probably higher than that provided for the average adult horse. For a diet containing 10% crude protein, the dietary BE might be of the order of 30 mEq/kg less.

Table 9.6 The effect of dietary BE on urinary and blood pH.

	Low	Medium	High
mEq/kg diet	22	202	357
Urinary pH	5.38	7.69	8.34
Blood pH	7.368	7.400	7.402

The absorption of Ca, Mg and P is limited and variable so that dietary cation anion balance (DCAB) is frequently measured in a simplified form as mEq/kg dietary DM:

$$\text{Dietary BE (mEq/kg)} = (\text{Na} + \text{K}) - (\text{Cl} + \text{S})$$

(NB: In some studies, the dietary S, normally providing 70–80 mEq/kg, is ignored.) On this basis, by including 10 g CaCl₂/kg in the 'medium' diet DM (Table 9.6) to give the 'low' diet and 13 g NaHCO₃/kg to give the 'high' diet, the urinary and blood pH were affected.

In the GI tract, H⁺ ions are exchanged for Ca²⁺ and Mg²⁺ ions by consumption of the 'low' diet. Urine is excreted in an electrically neutral state and, during exercise, immature horses given this diet could experience a considerable loss of urinary HCO₃⁻ and Ca²⁺ (hypercalciuria), accounting for metabolic acidosis and a negative Ca balance, demineralization and a weakening of the skeleton. Cooper *et al.* (1995) found raised urinary Ca²⁺ and Cl⁻ values in horses given a diet with a DCAB of -25.7 and relatively greater urinary losses of P, Na⁺ and K⁺ in horses given a diet with a DCAB of 370.4. Diets with a DCAB >200, as indicated above, reduce the risk of metabolic acidosis. Lower blood pH, PCO₂ and [HCO₃] values are found at rest (Baker *et al.* 1992) and subsequent to anaerobic exercise (Stutz *et al.* 1992) with a dietary (Na + K - Cl) balance of 5–21 cf. 107–125 or more mEq/kg. Horses receiving 107 mEq/kg, or less, recovered normal blood glucose more slowly after exercise than did those receiving 201 mEq/kg, or more. To maximize the transitory buffering effect of diet, the optimum time for anaerobic exercise is 3.5–4.5 hours after feeding (Stutz *et al.* 1992), despite an increased fluid load (Meyer 1992). Popplewell *et al.* (1993) recorded faster times over 1.64 km and lower heart rates after racing, two to four hours after a meal with a balance of 295 cf. 165 mEq/kg DM, despite higher blood lactates. Moreover, a balance of 354 mEq/kg DM achieved a greater Ca balance cf. 223 mEq/kg (Wall *et al.* 1993). The optima for balance and total mineral ion load is, however, likely to differ between types of work – sprint v. extended.

Dietary protein

The effects of dietary protein level on performance and metabolism during exercise are discussed in this chapter under 'Dietary protein requirements and exercise'. The effects of excess dietary protein, or that inevitably oxidized, on acid–base balance

have not been addressed. The effects may be small, but a basis for drawing conclusions should be summarized.

It must be remembered that only absorbed products of protein digestion can influence cation–anion balance. Many native proteins are rich sources of P, as phosphorylated amino acids, e.g. phosphoserine, and so contribute to the fixed anions. The oxidation of neutral amino acids has no effect on the acid load, but three classes of these nutrients do.

- (1) the basic (cationic) amino acids (lysine, arginine and histidine) yield neutral end products plus a proton (H^+) and thus are strangely acidogenic;
- (2) the sulphur-containing amino acids (methionine and cysteine) are also acidogenic, as they generate sulphuric acid when oxidized; and
- (3) the dicarboxylic amino acids (aspartic and glutamic acids) are anionic, but metabolizable, and consume protons when oxidized, and so reduce the diet's acid load.

Protons can be excreted in the urine but primarily as the ammonium ion ($H^+ + NH_3 \rightarrow NH_4^+$). Glutamine is the principal amino acid involved in renal ammonium ion genesis, and although not an essential dietary nutrient, it is used up in this process (see 'Protein assimilation', this chapter, glutamine drinks as an aid to recovery from prolonged exercise). Horse diets frequently have lysine and threonine as first and second dietary essential limiting amino acids and consequently there is interest in adding lysine-HCl to make good any limitation. When supplied as their Cl^- salts they are a fixed anionic, or acid, source. However, if 0.1%, or 1 g lysine-HCl/kg diet is added, it provides only 5.5 mEq acid/kg (See 'Dietary protein requirements and exercise' this chapter).

The effect on acid load of the rapid metabolism of amino acids during intense exercise may be important at the margin, but the amounts of acid produced are dwarfed by that produced through carbohydrate metabolism. Moreover, it is not established whether the rate of acid production from protein is markedly influenced by the dietary level of protein.

Effect of DCAB on digestibility and Ca and Mg retention

The cation–anion balance of diets has been artificially adjusted by additions of calcium chloride, ammonium chloride, potassium citrate and sodium bicarbonate, giving a range in balance of 20–400 mEq/kg diet. There is an increase in dry matter digestibility with an increase in positive balance. Hypocalciuria, promoting an increased retention of Ca and Mg, can also result from an increase in the balance, reducing the chronic risk of osteoporosis. A low dietary cation–anion balance induces metabolic acidosis, causes hypercalciuria, decreased Ca and Mg balances, and when horses are exercised within four hours of feeding, it leads to poorer work output and a slower recovery, owing to a poorer dietary buffering effect (Popplewell *et al.* 1993).

Supplements

Cofactors

The activity of enzymes depends on the presence of the necessary cofactors and an increasing demand for these enzymes implies an increasing need for the cofactors. These cofactors include magnesium and zinc together with forms of the vitamins thiamin, riboflavin, niacin, pantothenic acid, pyridoxine, biotin and vitamin B₁₂, all of which play major parts in carbohydrate and/or fat metabolism. The horse derives these B vitamins from its diet and by microbial synthesis in its intestine. As the intensity of work increases, the composition of the diet and the amount of feed consumed change as a consequence of the increased consumption of starchy cereal grains. This will alter not only the dietary supply of B vitamins, but also the intestinal synthesis of the vitamins, and it is an open question whether the rate of their absorption is exceeded by tissue demand when horses are in intensive training.

The microbial fermentation of starch cf. fibre yields a higher proportion of propionate in the VFA. Metabolism of this acid requires adenosylcobalamin (vitamin B₁₂), as the coenzyme of methylmalonyl-CoA mutase, and work with ruminants has revealed that such diets may create a dietary requirement for this vitamin (Agricultural Research Council 1980), the lack of which causes an accumulation of propionate, depressing appetite. Observations by the author (D. Frape unpublished observations) of horses in training have shown that their blood concentrations of vitamin B₁₂ are lower than in many other horses and that the palates of those with flagging appetites may be whetted by supplements of the vitamin. A reasonable inference is that an increased consumption of cereal grains by horses increases propionate production and hence the dietary requirement for vitamin B₁₂. An analogous argument may be put for thiamin, functioning as cocarboxylase in the cleavage of pyruvate. Studies by Topliff *et al.* (1981) suggested that the exercising horse may have a thiamin requirement double that of non-working horses. TBs in training have low serum folate concentrations, but whether this simply reflects a lower potency of training diets has not been established. Thus, there is no conclusive evidence concerning the effect of work on the requirement for B vitamins functioning as enzyme cofactors.

Amino acids

Branched-chain amino acids (BCAAs) stimulate protein synthesis for which growth hormone is a mediator. Oral supplementation of athletes with BCAAs (0.2 g/kg BW daily for one month) cf. controls led to a raised plasma concentration of growth-hormone-binding protein, slightly elevated growth hormone and lower plasma lactate concentrations (De Palo *et al.* 2001). I am unaware of any similar evidence in horses.

Water and electrolyte loading

Despite normal variation in the levels of each of the dietary electrolytes, adaptation serves to maintain the pH of body fluids in the normal physiological range. Outside this normal range, tissue pH may be altered through an overload of these compensatory mechanisms. Excretion of excess fixed ions requires water as a solvent, increasing water demand. Complete water restriction for 20 hours before exercise has been shown to reduce the intestinal water content by 10% at the start of protracted exercise, and that exercise reduces it by a further 15–20% through sweat losses, regardless of whether drinking had been allowed. Horses that are severely dehydrated are exceptionally exhausted (Carlson *et al.* 1976), and are reluctant to drink. Even during a ride, spontaneous drinking may not occur when water is offered if there has been an iso-osmotic loss in sweat, unless the fall in plasma volume exceeds 6% (Sufit *et al.* 1985) and especially if electrolytes are not given. Water and balanced electrolytes should therefore be given frequently during a ride.

Sodium chloride feeding before a ride could encourage water intake *then* and so should improve water balance during a subsequent extended ride. Loading the horse with electrolytes tends to increase their temporary accumulation, and that of water, in the large intestine (Slade 1987). This could act as a reserve of water, Na^+ (Meyer 1992) and of Cl^- (Coenen 1992a) for extended work. The daily ileocaecal flow of water and electrolytes per kilogram of body weight is in the range of 100–140 ml water, 300–420 mg Na^+ , 50–70 mg K^+ and 100–140 mg Cl^- . Absorption along with water from the large intestine, during ingesta fermentation, is estimated to be 75–95% for Na^+ , over 90% for Cl^- and 30–55% for K^+ (Meyer 1992). These nutrients can revive tissue depleted of water, Cl^- , Na^+ , K^+ and Ca^{2+} through sweating (Rose *et al.* 1977). The benefit of such an electrolyte reserve is promoted by the presence of fermentable material in the hind-gut, as this allows the continuous absorption of the reserve ions during a ride.

The amounts of dietary electrolytes necessary to keep a horse in electrolyte balance have been assessed to be 1.3–1.8 g Na, 3.1–3.9 g Cl, 4.5–5.9 g K, 8.5 mg Ca and 10.7 mg P/Mcal DE (0.3–0.4 g Na, 0.7–0.9 g Cl and 1.1–1.4 g K, 2 mg Ca and 2.6 mg P/MJ DE). Thus, the requirements for Na, K and Cl during exercise are increased over maintenance needs by three-, seven- and sixfold, respectively, according to Potter's group in Texas (Hoyt *et al.* 1995a). Natural feed, given after rides, is likely to contain much more K than Na. There should therefore be about twice as much Na as K and 1.2 times as much Cl as Na in supplements given with these feeds (Table 9.4). Organic anions can make up the residue. Small amounts of Ca and Mg may also be included.

Sodium bicarbonate

Anaerobic exercise causes a rise in plasma K^+ , released from the contracting muscle fibre. A failure of its reuptake may result from an inhibition of the Na–K pump of the muscle fibre membrane, owing to decreased ATP availability (Harris & Snow

1988) through inadequate buffering of H^+ ions within active fibres (Harris & Snow 1992). The loss of intracellular K^+ leads to an altered transmembrane potential that may contribute to fatigue during exercise.

To counter this, oral sodium bicarbonate ($NaHCO_3$), which increases plasma SID, results in a smaller rise in plasma NH_3 through a lower ATP loss and IMP formation (Greenhaff *et al.* 1991b), HCO_3^- accelerating H^+ removal (the bicarbonate system is the major proton acceptor in the body). A positive effect seems to occur only where the duration of exercise is 2–3 min, accounting for its particular use with Standardbreds engaged in races over 1.6–2.4 km. However, the use of alkalizing agents before a race is discouraged, or leads to disqualification, in many racing jurisdictions. No effect of $NaHCO_3$ was observed over 1 km (Greenhaff *et al.* 1991b). Lawrence *et al.* (1987a, 1990) reduced race times by 1.1 s over 1.61 km with Standardbreds treated orally with 0.3 g/kg BW mixed with 20 ml corn syrup and 10 ml water, cf. powdered confectioner's dextrose and salt mixed with corn syrup and water, 2.5 hours before exercise ($P < 0.1$). Treatment increased both blood pH and lactate disappearance rate after racing. Harkins & Kamerling (1992) treated TBs with 0.4 g $NaHCO_3$ /kg BW in 1 l water, cf. 1 l water only, three hours before a 1.61 km race, increasing venous HCO_3^- and pH. After the race, there was an increase in venous blood pH and lactate in the $NaHCO_3$ group, with no change in race times, or in venous partial pressure of carbon dioxide (venous $vPCO_2$). (Note that the lactate⁻ ion [Lac^-] will be neutralized by Na^+ and it does not determine the pH. Alternatively, it can be argued that the increase in [Na^+] reduces plasma [H^+] through the maintenance of electroneutrality, despite an increase in plasma [Lac^-]. This increase in [Lac^-] is probably the result of increased efflux of Lac^- from muscle cells caused by the extracellular alkalosis, so reducing fatigue.) The optimum dose and time are 0.4 g $NaHCO_3$ /kg BW (in 1 l water) two to four hours prior to work, as assessed by blood pH and HCO_3^- (Greenhaff *et al.* 1990b; Corn *et al.* 1993), although doses of 1 g/kg BW have achieved higher values with a peak four hours after administration.

Analysis of venous blood from Standardbred pacers before racing has revealed HCO_3^- values in excess of 40 mmol/l, indicating higher doses than 0.4 g/kg. A dose of Na equivalent to 20% of the body's total exchangeable Na should increase plasma volume, which could have an opposite effect on sprint performance to that of a buffer, and may account for variable results following $NaHCO_3$ administration. Moreover, the value of the large intestine as a source of Na may be modulated by acetate production (Argenzio *et al.* 1977), which varies with the interval from, and nature of, the last meal. Lloyd *et al.* (1993) administered 1 g $NaHCO_3$ /kg BW, cf. a similar molar dose of NaCl or water only. The $NaHCO_3$ extended exercise to exhaustion on a treadmill and increased blood lactate, but a comparison with the two untreated groups, where all horses had access to water, indicated poorer endurance with $NaHCO_3$, possibly from a higher fluid load. Yet Hanson *et al.* (1993) gave horses, with and without free access to water, 1 g $NaHCO_3$ /kg BW in 4 l water and found no significant difference in plasma volume. The response is, however, more complicated, as alkalosis, caused by the 1 g dose/kg, led in both studies

to hypercapnia and some hypoxaemia through respiratory compensation. This ventilatory depression was not thought to have affected performance, and was probably associated with reduced tidal volume because of the tendency for a 1:1 linkage of respiratory frequency with stride. Moreover, intracellular $[H^+]$ may exchange for extracellular K^+ , causing hypokalaemia after administration of 'milk shakes' of sodium bicarbonate. Both plasma K^+ and Ca^{2+} concentrations decline with alkalosis so that cardiac and skeletal muscle contractions could be disrupted. This may contribute after exercise to the distress of synchronous diaphragmatic flutter, and other signs.

Thus, the optimum dose, method of administration and overall effects of $NaHCO_3$ and water have yet to be determined. Nevertheless, some value may result from treatment with 0.4 g $NaHCO_3$ /kg BW (in 1 l water) two to four hours prior to gallops lasting 2–3 min.

Calcium carbonate and sodium chloride

Frey *et al.* (2001) compared iso-osmolar amounts of sodium bicarbonate (500 mg/kg BW) and calcium carbonate (595 mg/kg BW) with water, and demonstrated that calcium carbonate had no effect on blood pH or bicarbonate $[HCO_3^-]$ level. On the other hand, 488 mg sodium acetate/kg BW (pKa 4.8) produces a similar degree of metabolic alkalosis to an isocationic dose of 500 mg sodium bicarbonate/kg BW (pKa 6.1), when they are administered three hours before a fast mile (Frey *et al.* 1999). By comparison, it was previously noted that sodium chloride decreases arterial blood pH, inducing slight acidosis (Hinchcliff *et al.* 1993).

Carnosine

A more enlightened approach to combating the rise in intracellular $[H^+]$ may be to alter the intracellular concentration of the imidazole dipeptide buffers carnosine (β -alanylhistidine) and its N^2 -methyl derivative, anserine. Carnosine contributes 30% of the buffering in equine skeletal muscle (Harris *et al.* 1991a), and, in type IIb fibres (prominent in equine muscle), it may account for up to 50%, with a concentration of 188 mmol/kg DM muscle (Sewell *et al.* 1991a,b; Sewell 1992b). However, dietary supplements of histidine have not given convincing responses in delaying fatigue.

FAT SUPPLEMENTS AND EXERCISE

Horses participating in competitive long-distance rides are required to make effective use of body-fat reserves as a source of energy to conserve glucose sources, as a severe depression in blood glucose is an indication of fatigue. Good-quality dietary oils, fats and medium-chain triacylglycerols are all well utilized (McCann *et al.* 1987; Hollands & Cuddeford 1992; Potter *et al.* 1992b; Jackson *et al.* 2001). MCTs have carbon-chain lengths 6–12 and are rapidly absorbed, followed by portal transport to

the liver. They are independent of mitochondrial transport enzymes and, with hepatic metabolism to ketones, are oxidized in the presence of O_2 . Vegetable fats are, however, generally more readily digested than fats from animal sources.

Fats given to man and to the horse seemingly delay gastric emptying of carbohydrate, and so improve glucose tolerance, by lowering the post-meal peak plasma glucose response. Fats are not subject to microbial fermentation and their greater use decreases colic and laminitis risks and may promote intramuscular and hepatic fat catabolism, increasing performance at submaximal and intense rates. Nevertheless, the rate of endogenous *de novo* fatty-acid synthesis may be reduced. A diet supplemented with soya-bean oil reduced fasting plasma triacylglycerol concentrations (Orme *et al.* 1997; Geelen *et al.* 2001). The authors concluded that, at least in part, this was caused by a reduction in fatty-acid synthesis, indicated by decreased activities of hepatic acetyl-CoA carboxylase (ACC, EC 6.4.1.2) and fatty acid synthase (FAS) activities.

Work in Texas with Quarter Horses used for cutting shows that the benefits of a 10% fat supplement are adaptive and take 3–4 weeks to materialize (Julen *et al.* 1995). Fat supplements can delay the decline in blood glucose during endurance rides, accelerate the rate of recovery of resting pulse and respiration rates (Hintz *et al.* 1978a,b; White *et al.* 1978) and promote the recovery of resting blood glucose, reducing the risk of fatigue-related injuries. However, they can also reduce concentrate and total feed intake of exercised horses (Besancon *et al.* 1999) and practical problems of adding large amounts of fat to the diet have to be addressed.

Fat yields less CO_2 /mole ATP generated, decreasing plasma PCO_2 . Therefore, relative to the effects of a carbohydrate diet, there is a small increase in plasma pH brought about by a fat supplement in exercised horses with decreases in heart rate, plasma $[H^+]$ and lactate concentration, and so an increase in venous SID, with a delay in fatigue (Kennedy *et al.* 1999; Taylor *et al.* 1999). Hard training (Hambleton *et al.* 1980) and fat supplementation with anaerobic (Pagan *et al.* 1993b) and extended aerobic (Pagan *et al.* 1987c) exercise are followed by an elevation in plasma FFAs, whereas resting FFAs are lowered by supplementation (Harkins *et al.* 1992). Thus, there may be a stimulation to β -oxidation, or to both fat mobilization and metabolism (Figs 9.15 and 9.16), sparing glycogen. In this way, triacylglycerols do not accumulate in plasma (Kennedy *et al.* 1999). These responses result from increases in muscle lipoprotein lipase (LPL) activity and in citrate production which inhibits phosphofructokinase, one of the rate-limiting enzymes of glycolysis. This in turn results in an accumulation of glucose-6-phosphate, inhibiting glucose phosphorylation and sparing glucose oxidation.

Evidence of Orme *et al.* (1997) and Geelen *et al.* (2001) indicated that while hepatic acetyl-CoA carboxylase and fatty acid synthase activities were reduced, fat supplementation raised plasma total lipase (following pentosanpolysulphate administration), skeletal muscle citrate synthase (EC 4.1.3.7) and carnitine palmitoyltransferase-I (EC 2.3.1.21) activities. The authors concluded that post-prandial plasma TAG clearance was promoted and both transport of fatty acids through the mitochondrial inner membrane and the oxidative capacity of highly

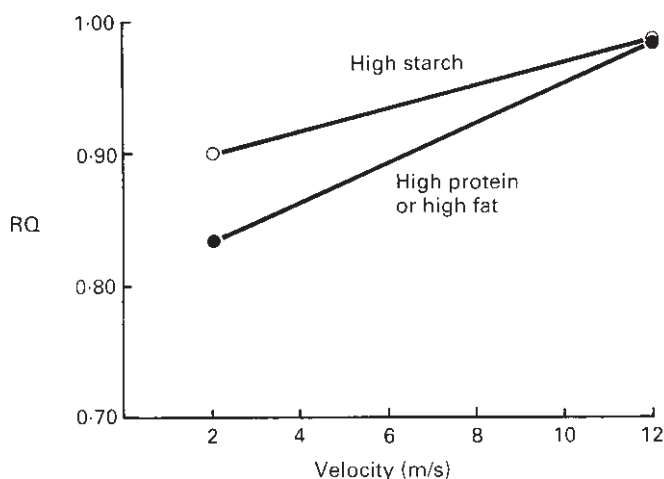


Fig. 9.15 Relationship between RQ and velocity of horses given high starch, high protein or high fat diets. At high velocity there is no difference in RQ because high rate of energy expenditure demands glycolysis. At low to moderate velocity, fat and protein may be used (Frape 1994).

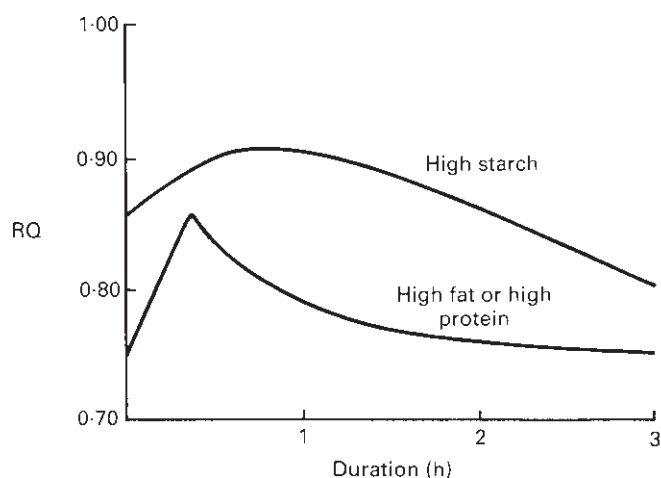


Fig. 9.16 Relationship between RQ and time during aerobic work (low to moderate velocity, 4–6 m/s) in horses given high starch, high fat or high protein diets. On high fat or high protein diets, fat mobilization becomes predominant earlier, thereby conserving muscle glycogen (Frape 1994).

aerobic muscles were enhanced by fat feeding. The addition of 100g corn oil to the diet of sprint-trained Arabian horses (Taylor *et al.* 1993, 1995) caused, at fatigue, greater plasma glucose and lactate⁻ (11 v. 8mEq/l) concentrations, offset by increases in plasma [Na⁺] and [K⁺] and decreases in plasma [Cl⁻]. Thus, the fat raised SID, minimized the decrease in pH of plasma and it may reduce PCV in exercised horses in hot weather, possibly indicating greater reserves of water in the extracellular fluid (Mathiason-Kochan *et al.* 2001).

Table 9.7 Blood [Lac⁻] during sprints in horses adapted to either a control or high-fat diet and administered either water or NaHCO₃ before exercise (Ferrante *et al.* 1994b).

	Blood [Lac ⁻] (mmol/l)
Control/water	5.73 ± 0.12
Control/NaHCO ₃	6.26 ± 0.21
Fat/water	7.01 ± 0.20
Fat/NaHCO ₃	9.47 ± 0.32

Three points to note concerning fat supplementation are:

- (1) increased plasma lactate may possibly cause fatigue independently of an effect on plasma pH;
- (2) the increase in lactate in Arabians is less than that observed in TBs. This may be associated with the higher proportion of slow-twitch, oxidative muscle fibres (type I), and greater oxidative enzyme activity in Arabians; and
- (3) the greater rise, after exercise, in plasma lactate (and alanine, a precursor of pyruvate and of glucose) with fat supplementation possibly results from the accelerated rate of glycogenolysis coupled with a reduction in the activity of pyruvate dehydrogenase (PDH) complex and decreased oxidation of pyruvate. PDH is a key regulator of fat and carbohydrate metabolism (Fig. 9.11). There can in fact be a synergistic effect of the combined treatment with fat and NaHCO₃, leading to even higher plasma lactate levels (Table 9.7). There is a lower rate of pyruvate conversion to acetyl-CoA, as β -oxidation increases production of acetyl-CoA. However, much evidence in horses run at a constant velocity indicates a lower plasma lactate accumulation with dietary fat supplements (Table 9.10). This points to a substitution of β -oxidation for glycogenolysis, conserving glycogen stores.

During sprinting, glycogenolysis is required as an anaerobic high-power source to complement the greater oxidation of fatty acids, a low-power source, associated with fat supplementation. Although plasma lactate may be increased, there may be no significant decrease in plasma pH, or SID, if there are increases in plasma Na⁺ and K⁺ concentrations and a decrease in plasma Cl⁻ concentration.

Heat increment

The increased energy density and decreased intestinal residue achievable with dietary fat supplements could be the essential characteristic of fat (Hiney & Potter 1996). When 10% fat replaced starch, heat production fell from 77% of available DE to 66% and available NE rose from 16% of DE to 36% during work (Scott *et al.* 1993), reducing thermal stress (McCann *et al.* 1987), regardless of body fatness and

in both temperate and hot weather (Potter *et al.* 1990). Waste heat production during exercise is very large. Approximately 80% of stored energy utilized for movement is lost as heat. The decrease in heat production achieved by fat supplementation in the main reflects diminished microbial fermentation in the hind-gut.

Muscle glycogen

Several reports indicate no difference in resting muscle glycogen concentration between high starch and high fat diets (Hintz *et al.* 1978a; Pagan *et al.* 1987a; Topliff *et al.* 1987; Orme *et al.* 1997). Both Pagan *et al.* (1987b) and Geelen *et al.* (2001), equalizing DE intake between treatments, and Greiwe-Crandell *et al.* (1989) providing higher energy intakes with fat supplements, found lower concentrations. Most other reports describe increased resting (post-exercise muscle glycogen concentration may be no higher as glycogen utilization may be promoted by its higher muscle level) muscle glycogen following vegetable, or animal, fat additions at approximately 10% of the diet, to provide equal DE or ME intakes (Hambleton *et al.* 1980; Meyers *et al.* 1987, 1989; Oldham *et al.* 1990; Harkins *et al.* 1992; Jones *et al.* 1992; Scott *et al.* 1992; Julen *et al.* 1995) (Table 9.8).

Effects of fat on hepatic glycogen capacity, which is 10% of that in skeletal muscle, are equivocal, as marginal increases (Hambleton *et al.* 1980) and decreases (Pagan *et al.* 1987b) are reported. Although fat concentrations of up to 20% of the dietary DM have been used without digestive upset, or any reduction in utilization, dietary fat levels of 15% have decreased glycogen storage compared with controls.

Table 9.8 Resting muscle glycogen¹ as affected by added dietary fat in diets of differing energy densities but generally given to horses to equalize DE intake (Frape 1994).

Added dietary fat (g/kg diet)	0	20–30	50–60	80–100	140–150	SE	Reference
Muscle glycogen (mmol/kg wet tissue)							
Animal fat	81	—	—	78	—	—	Hintz <i>et al.</i> 1978a
	94	—	109	143	—	10.5	Meyers <i>et al.</i> 1989
	88	—	—	127	—	2.6	Oldham <i>et al.</i> 1990
	93	—	—	145	—	2.1	Scott <i>et al.</i> 1992
Muscle glycogen (mmol/kg DM)							
Vegetable oil ^{2,3}	—	200	255	292	240	—	Hambleton <i>et al.</i> 1980
	680	—	—	—	580	100	Pagan <i>et al.</i> 1987a
	198	229	—	—	—	12	Harkins <i>et al.</i> 1992

¹ *Gluteus medius*, *biceps femoris* or *quadriceps femoris*.

² Fat-added diet replaced maize grain giving diets of different energy densities but constant daily energy and protein intakes.

³ Fat-added diet contained less roughage but more fat, starch and protein and was fed to provide equal DE intakes.

A diet clearly needs to contain enough starch from which the storage is derived. Meyer & Sallmann (1996) found that when 2 g fat/kg BW daily was given, as much as 0.4 g fat/kg BW was transferred to the hind-gut, with a potential risk for disturbance of caecal microbial metabolism. Hence, an optimum dietary concentration of approximately 100 g/kg exists.

Respiratory quotient (RQ)

Respiratory quotient is the ratio at standard temperature and pressure (STP) of the volume, or moles, of CO₂ eliminated, to the volume, or moles, of O₂ utilized, in oxidation, i.e. CO₂/O₂ for carbohydrate = 1, fat = approx. 0.7 and amino acids = approx. 0.85. RQ rises with increasing speed (Pagan *et al.* 1987b), is lowered by training (Meyers *et al.* 1987) and is either not affected by dietary fat (Meyers *et al.* 1989) or is lowered by additional protein or fat during submaximal exercise (Pagan *et al.* 1987b) (Table 9.9; Figs 9.15 and 9.16). A lower RQ implies a lower rate of CO₂ production.

A decreased PCO₂ may moderate a decrease in blood pH (through maintenance of the dissociation equilibrium of carbonic acid), so offsetting fatigue. RQ is positively correlated with muscle glycogen reserves during mild aerobic exercise and it declines as submaximal exercise progresses (Pagan *et al.* 1987b) (Fig. 9.16), indicating a sparing of glycogen. Higher stores of glycogen, with fat supplementation, accelerate their mobilization rate during anaerobic exercise (Oldham *et al.* 1990; Jones *et al.* 1992; Scott *et al.* 1992; Julen *et al.* 1995). Yet a lower rate (Greiwe-Crandell *et al.* 1989), no difference (Hintz *et al.* 1978a) and a marginally greater loss of glycogen, with lower initial reserves (Pagan *et al.* 1987b), have all been reported during aerobic exercise with fat supplements (Figs 9.17 and 9.18).

No clear picture emerges that fat would particularly benefit exercise in which extended aerobic metabolism dominated. Metabolic adaptation to a fatty diet may take as much as 6–11 weeks (Custalow *et al.* 1993) and some studies may not have allowed sufficient time for this, so that their outcome could have depended on

Table 9.9 Relationship of added dietary fat to RQ in horses exercised on treadmills (Frape 1994).

Speed (m/min)	Slope (°)	Time (min)	Added dietary fat (g/kg diet) (SE)				Reference
			0	50	100	150	
					R	Q	
180	9	20	0.910	0.860	0.870	—	Meyers <i>et al.</i> 1989
300	0	90	0.830 (0.012)	—	—	0.750 (0.022)	Pagan <i>et al.</i> 1987b
360*	0	2	0.887 (0.019)	—	—	0.827 (0.038)	Pagan <i>et al.</i> 1987b
480*	0	2	0.890 (0.031)	—	—	0.873 (0.030)	Pagan <i>et al.</i> 1987b
				—	—		
600*	0	2	0.977 (0.027)	—	—	0.957 (0.043)	Pagan <i>et al.</i> 1987b

*Step-wise work test.

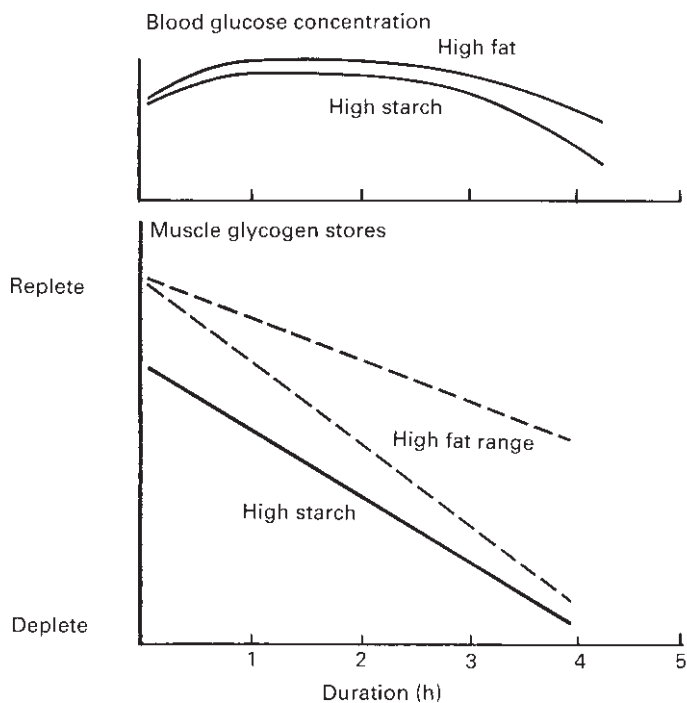


Fig. 9.17 Generalized relationships, with time, of blood glucose and muscle glycogen concentration in horses of moderate fatness during extended aerobic work (Frape 1994).

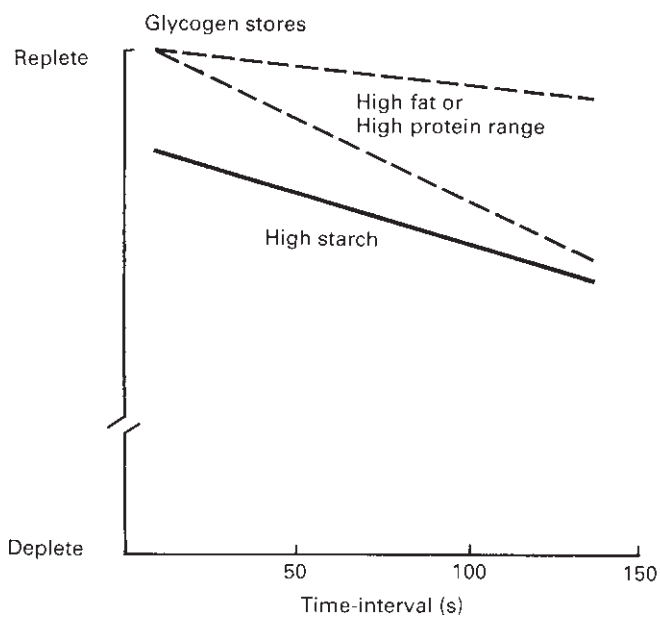


Fig. 9.18 Generalized relationship between glycogen stores and time-interval during intense anaerobic work (>600 m/min, >190 beats/min) (Frape 1994).

design details and horse temperament. The interpretation is complicated by the expression of harder anaerobic work (Webb *et al.* 1987a) and faster speeds, with no greater glycogen loss, at a constant heart rate (Oldham *et al.* 1990) by fat supplemented horses. As fat yields energy only by oxidation, minimal glycogen sparing would be expected during maximal exertion, when its value is other than for conservation of glycogen.

Blood glucose

Some workers report similar (Worth *et al.* 1987) or lower (Meyers *et al.* 1989) blood glucose concentrations with added fat during aerobic exercise, but the majority (Hintz *et al.* 1978a; Hambleton *et al.* 1980; Webb *et al.* 1987a; Oldham *et al.* 1990; Harkins *et al.* 1992; Scott *et al.* 1992; Custalow *et al.* 1993) observed higher values during and after exercise of all types (Fig. 9.17 and Table 9.10), even with increased work effort (Webb *et al.* 1987a; Harkins *et al.* 1992).

Lower heart rates and a more rapid recovery of resting rates (Meyers *et al.* 1987), lower blood lactic acid concentrations during and subsequent to submaximal and strenuous standardized exercise tests (SETs) in fat-supplemented horses (Pagan *et al.* 1987a,c; Webb *et al.* 1987a; Meyers *et al.* 1989; Pagan *et al.* 1993b) (Table 9.10), a higher velocity (m/s) at which venous blood lactate attains 4 mmol/l ($\dot{V}_{LA4}$) (Pagan *et al.* 1993b) and lactate speed threshold (Custalow *et al.* 1993) possibly reflect a slightly lower RQ (Table 9.9 and Fig. 9.15), but an absence of blood pH measurements complicates fatigue assessment. Higher lactates during some higher speed SETs (Ferrante *et al.* 1993; Taylor *et al.* 1993) with fat cf. starch may represent a tendency for the response curves to crossover at these speeds (Custalow *et al.* 1993).

Table 9.10 Effect of fat supplementation on blood plasma lactate and glucose concentrations during exercise at constant velocity (C) or at uncontrolled velocity (UC) and after post-exercise rest. Data averaged over the sources used for each comparison of carbohydrate control and added fat (Frape 1994).

Added dietary fat (g/kg)		Plasma lactate (mmol/l)		Plasma glucose (mmol/l)		Reference
		Work	Rest	Work	Rest	
0	C	2.30	1.98	5.42	5.32	Hambleton <i>et al.</i> 1980; Meyers <i>et al.</i> 1987, 1989; Webb <i>et al.</i> 1987a; Worth <i>et al.</i> 1987
100	C	1.89	1.47	4.93	5.23	
0	C	9.90	—	2.52	3.80	Pagan <i>et al.</i> 1987c
100	C	6.70	—	2.82	3.70	
0	UC	2.31	2.25	5.32	6.02	Hintz <i>et al.</i> 1978a; Harkins <i>et al.</i> 1992
100	UC	2.12	2.09	6.11	6.25	
0	UC	15.79	2.76*	6.57	5.29*	Webb <i>et al.</i> 1987a; Oldham <i>et al.</i> 1990 Scott <i>et al.</i> 1992
100	UC	18.61	1.87*	7.05	6.02*	

*Data from Webb *et al.* 1987a only.

The effects of high protein on blood lactic acid may be more prominent than those of high fat (Pagan *et al.* 1987a,c). Observations in human subjects indicate that high protein/high fat diets increase the activity of skeletal muscle lipoprotein lipase (LPL), whereas high carbohydrate diets reduce that activity (Jacobs 1981). As the insulin response to a carbohydrate diet exceeds that to high protein/high fat diets, and as insulin depresses muscle LPL activity, the observation of Jacobs is understandable. Increased energy generation from fat oxidation with high fat and protein diets may owe to the combined effects of increased muscle LPL hydrolysis of plasma TAG and increased use of plasma FFA (Figs 9.16 and 9.17), accounting for lower plasma lipids during aerobic SETs (Meyers *et al.* 1987). No certain conclusions can, however, be drawn from changes in venous blood FFA values (Frape 1993), but it is suggested that high fat diets may increase activity of muscle LPL (and possibly of TAG lipase), reciprocally reducing adipose tissue LPL activity, in contrast to the effects of starch (Fig. 9.11). As plasma TAG concentration tends to decline with fat supplementation, the increase in muscle LPL activity would seem to exceed the reciprocal decrease in adipose tissue LPL activity.

A dietary increase in either protein or fat normally results in decreased starch, reducing 'heating', anxiety, heart rate and excitability. Mixed fats contain lecithins, a component of which is choline. Holland *et al.* (1996) reported that vegetable oil, or especially oil enriched with additional soya lecithin, reduced the spontaneous activity and excitability of horses when the diet contained 100 g of this supplementary oil/kg. Choline is used in the synthesis of acetylcholine, a neurotransmitter found in parasympathetic nerve synapses and in voluntary nerves to skeletal muscles. The potential advantages and disadvantages of fat are proposed in Table 9.11 (also see 'Polyunsaturated fatty acids', Chapter 5).

Polyunsaturated fatty acids and 3-thiobarbituric acid reactive substances (TBARs)

Linoleic acid is a dietary essential polyunsaturated fatty acid (PUFA). Oils especially rich in PUFA have no notable benefit for ponies given a diet deficient in PUFA for seven months (Sallmann *et al.* 1992). However, chain length, or degree of unsaturation, may influence the exercise response (Pagan *et al.* 1993b). PUFAs in cell membranes are susceptible to attack, with the removal of an H atom with its electron, leaving a radical subject to attack by O₂, yielding peroxy radicals. Chain reactions, the breakdown of the cell membranes and several products that include malonyldialdehyde (MDA), n-pentane and ethane are a consequence. MDA can be measured colorimetrically with thiobarbituric acid (TBA). Strenuous exercise causes increased plasma thiobarbituric acid reactive substances (TBARs) and breath n-pentane per kilogram of body weight (McMeniman & Hintz 1992). However, the peroxidative stress of 3% corn oil was accommodated in exercising ponies given 42 iu vitamin E/kg dietary DM, through increased plasma glutathione peroxidase and superoxide dismutase activities and increased ascorbic acid concentration, despite higher muscle TBARs and regardless of vitamin E concentration.

Table 9.11 Provisional conclusions on effects of high fat diets given to exercising horses compared with diets of normal fat concentration providing similar amounts of dietary fibre, protein and DE but more starch (Frape 1994).

Advantages

- (1) Lower RQ during submaximal exercise (promoting fat catabolism) potentially extending endurance.
- (2) Possible decrease in adipose tissue LPL activity (EC 3.1.1.34) and increase in muscle LPL activity.
- (3) Increase in muscle glycogen stores, more glycolytic energy and possible delay in glycogen exhaustion during extended aerobic exercise.
- (4) Increased, or sustained, blood glucose concentrations during extended exercise.
- (5) Possibly delayed lactic-acid accumulation during anaerobic exercise (lactic-acid accumulation is proportional to the rate of glycogen expenditure, when other conditions are constant).
- (6) Reduction in gut fill, which may benefit work at >200 beats/min, but which may compromise endurance.
- (7) Reduced excitability of hot-blooded horses and a possible reduction in risks of colic and laminitis in all types of horse.
- (8) Fats contain lecithins, a component of which is choline which is a precursor of acetylcholine, the neurotransmitter. Lecithin feeding is associated with reduced excitability.

Disadvantages

- (1) High cost of high-grade fat.
 - (2) Wide availability of poor-quality feed-grade fat and difficulty of assessing quality.
 - (3) Lack of stability of large fat supplements in mixed feed and practical problems of administration to the horse.
 - (4) Refusal of high-fat diets, or delay in acceptance of equivalent intakes, i.e. lower palatability.
 - (5) Lower large intestinal fluid reserves for endurance events.
-

DIETARY PROTEIN REQUIREMENTS AND EXERCISE

A number of investigations have demonstrated that dietary protein concentration can influence running ability in horses. However, it is estimated that protein catabolism accounts for no more than 5–15% of the energy consumed during exercise, yet in both extended work (Rose *et al.* 1980) and exercise in the post-absorptive state following high protein meals (Miller-Graber & Lawrence 1988), plasma urea is elevated. The NRC (1989) has concluded that dietary protein requirements of horses are proportional to those for DE.

Protein assimilation

Protein assimilation of tissue occurs during work following rest (Meyer 1987), but its scale is unclear. Johnson *et al.* (1988) detected no increase in nitrogen (N) balance of working ponies. Patterson *et al.* (1985) found that 1.9 g digestible protein/kg BW^{0.75}, equivalent to 5.5% of dietary protein (maize soya protein), was adequate for intense exercise (cf. 7% and 8.5% dietary protein), as measured by plasma total protein, albumin and urea N. Orton *et al.* (1985a) trotted growing horses for 12 km daily at 12 km/hour, on either a 12–14% or a 6–8% protein diet. Exercise increased feed and protein intakes, and consequently growth rate, of horses on the low protein

diet to equal that of those receiving more protein. The greater appetite provided protein, surplus to exercise requirements, utilized for growth. In a survey of racing TBs, Glade (1983a) found that protein intake (confounded with DE intake) was positively correlated with time to finish, implying that excess protein depressed speed. It is concluded that there is little justification for greatly increasing the daily protein intake of exercising horses to meet some putative increase in chronic requirement.

The relationship between dietary protein and extreme performance is, however, far from clear. Despite seemingly high NRC (1989) estimates, actual intakes are still higher. Yet an excess of 56% over the estimates among racing Standardbreds (Gallagher *et al.* 1992a), and of 21% for racing TBs (Gallagher *et al.* 1992b) may reflect the natural protein content of palatable high-energy feeds. RQ was lower in horses given a high protein, cf. control, diet and exercised at high speed (Pagan *et al.* 1987b), implying a stimulation to protein or fat metabolism in the post-absorptive state (Fig. 9.15), increasing urea yield (Frank *et al.* 1987) and water needs. Apart from this increased need, Hintz *et al.* (1980) observed no detrimental protein effect in horses during distance riding, where dehydration causes fatigue. Miller-Graber & Lawrence (1988) recorded a higher plasma urea N, 16–19 hours after an 18.5% cf. 12.9% protein meal during 15 min work at 170–180 beats/min; but plasma NH_3 rose to the same extent in both groups, jugular lactate concentration increased less, the increase in plasma glutamine was marginally less and that of plasma alanine significantly less in the high protein group ($P < 0.05$). Others have also observed lower plasma lactic-acid concentrations during intense (Pagan *et al.* 1987b,c) and less intense (Frank *et al.* 1987) exercise with high protein diets (24.6% and 20%, respectively, v. 14.6% and 10% protein, respectively), decreasing heart rate and glycogen catabolism at high speed (Pagan *et al.* 1987b). High protein reduced the post-exercise rise in plasma NH_3 only in untrained (Frank *et al.* 1987) and not trained (Miller-Graber & Lawrence 1988) horses. Observations in the fasting state may have excluded an adverse excess protein effect. Thus, Miller-Graber *et al.* (1991) performed the test three to four hours after a meal, when 9% cf. 18.5% dietary protein was inconsequential for hepatic, or intramuscular, glycogen use, or for venous blood lactate concentration. Nevertheless, five minutes after exercise, venous blood lactate:pyruvate ratio was higher with the 9% diet, possibly indicating a higher pyruvate dehydrogenase (EC 1.2.4.1) activity, or decreased NADH:NAD ratio, with that diet.

It is concluded that high protein diets, above the need for N balance, may confer some metabolic advantages to working horses (Table 9.12 and Figs 9.11, 9.15, 9.16 and 9.18). Nevertheless, excess protein is oxidized, resulting in the production of urea, heat and acid. Graham-Thiers *et al.* (2001) compared diets containing 7.5% and 12% protein, but with a similar DCAD (meq/kg, $\text{Na} + \text{K} - \text{Cl} - \text{S}$). At rest the higher protein diet led to a lower blood pH, owing to a difference in SID ($\text{Na}^+ + \text{K}^+ - \text{Cl}^- - \text{lactate}^-$). Both exercise and a higher protein diet can contribute to acidosis, so such a diet may compromise acid–base balance during exercise, in addition to creating additional waste heat. Moreover, increased urea production in the stable

Table 9.12 Some metabolic responses of untrained, exercised horses to dietary protein intakes well in excess of N balance, compared with responses at approximately N balance with diets providing similar amounts of starch.

-
- Increase in blood urea concentration.
 - Decrease in venous blood ammonia concentration after exercise.
 - Decrease in RQ during aerobic exercise.
 - In the range 240–600 m/min
 - decrease in venous blood lactate concentration.
 - decrease in hepatic lactate concentration.
 - decrease in venous blood, lactate:pyruvate, ratio.
-

could increase environmental ammonia, contributing to respiratory stress. (It is of current interest that glutamine drinks, taken after exercise by long-distance human athletes, reduce the frequency of respiratory infections. The reason seems to be that during extreme exertion glutamine sources are depleted and glutamine is an essential fuel for the functioning of the immune system – the equine possibilities have not been examined, to the author's knowledge.)

Specific amino acids

There is an increase in plasma free lysine and phenylalanine after exercise. As these amino acids are not normally catabolized for energy, their increase indicates an increase in net protein catabolism during exercise. Even so a 7.5% protein diet supplemented with 5 g/kg lysine and 3 g/kg threonine, cf. a 14% protein diet (low- and high-protein diets contained totals of, respectively 6.1 g and 6.9 g L-lysine and 5.2 g and 5.9 g L-threonine per kg) led to lower plasma and urinary urea and uric acid, similar plasma albumin levels and a moderated acid–base response to sprints when compared with the effects of the high diet in exercising Arabians (Graham-Thiers *et al.* 1999, 2000). Higher plasma creatinine concentrations with the lower protein diet indicated to the authors that the amino acid supplement may have supported additional muscling. Valine and isoleucine are glycogenic and readily oxidized to provide energy. Supplements, including these branched-chain amino acids, have resulted in a lower plasma lactate accumulation and lower heart rates, when given 30 min before exercise.

FEEDING METHODS

Feed sequence, protein utilization and plasma amino acids

The true digestibility of protein in the equine small intestine ranges from 45% to 80%. At high rates of protein intake more will be degraded to NH_3 in the large intestine. Utilization of this by gut bacteria is 80–100% (Potter *et al.* 1992c). Excessive protein intakes must inevitably increase the burden of unusable N either in the

form of inorganic N or as relatively unusable bacterial protein. This burden is influenced by feeding sequence. The provision of a concentrate feed two hours after roughage, cf. simultaneous feeding, caused higher levels of plasma free, and particularly essential, amino acids, six and nine hours later, respectively (Cabrera *et al.* 1992), indicating improved nutritional value derived from delaying the concentrate. Plasma urea did not rise with this dissociated feeding, but it rose continuously for the nine hours after the mixed feeding, implying there was a large caecal flow of digesta.

Protein and heat production

Belko *et al.* (1986) found that the thermic effect of food in exercising men increased with the protein content 150–270 min postprandially. No similar measurements of heat production in horses are available, although Frank *et al.* (1987) measured no difference in heart rate, or body temperature, between horses trained on diets containing 10% and 20% protein. However, the feeding sequence referred to above may influence heat production, as deamination and urea synthesis are associated with additional waste heat. The optimal amount and preferred feeding practice for dietary protein are thus not established, but optima for both may exist for both intense and extended exercise. The French evidence (Cabrera *et al.* 1992) may suggest that the feeding sequence should be the reverse of normal practice. Improved husbandry may allow the achievement of maximal effects without a burden of amino acid degradation products. Deamination of AMP is clearly prominent during brief maximal exercise (Miller-Graber *et al.* 1987) (Fig. 9.11). Alanine is the principal vehicle for shuttling NH_3 from muscles to liver, but whether this is stimulated by extra protein given during rest is not definitely established.

Processing of cereals and precaecal digestion

The extent to which cereal starch provides glucose, or VFAs, depends upon its precaecal and even its preileal digestibility. Kienzle *et al.* (1992) reported that the preileal digestibility of oat starch was higher than that of maize starch, with similar degrees of processing. Grinding of whole grain led to high preileal digestibility (%), for oats amounting to 98.1% and for maize 70.6%, while rolling, or breaking, had little effect (whole oats 83.5%, rolled oats 85.2%, whole maize 28.95%, broken maize 29.9%). Starch gelatinization enhances its small-intestinal digestion, but at moderate, or high, rates of intake only. At low rates (<0.4% of body weight per meal) most sources of starch are digested in the small intestine (Potter *et al.* 1992a). Thus, end products, gut fill and possibly the optimum time interval between feeding and exercise are all influenced. A change in the proportions of fatty acids to glucose in the end products, which can be influenced by processing, may modulate exercise performance (Frape 1994).

'Hotting-up' and the heat increment of feed

In Chapter 6 the phenomenon of waste heat generated during the digestion and metabolism of feeds was outlined and a mechanism for the 'hotting-up' effects of certain feeds was adduced earlier in this chapter ('Fat supplements and exercise'). Many trainers and horsemen and -women are reluctant to use energy-rich cereals such as maize and barley because of the alleged risks in this connection. However, the explanation here makes clear that where alternative feeds are rationed at rates that provide the same amounts of net energy, then energy-rich cereals will generate *less* rather than *more total* heat over a period of 12 hours, or more. Measurements in polo ponies maintained at a constant body weight support the conclusion that energy-rich feeds do not necessarily exacerbate a heating effect of feed, but body temperature and metabolic rate increase after feeding (Fig. 9.19). The ponies received approximately equal amounts of net energy from either maize and lucerne hay, or oats and timothy hay. No significant differences in response owing to diet, either before or after exercise, were noted (Wiltzie & Hintz in Hintz 1983). If anything, the maize-fed animals were less 'hotted-up'. Energy-rich feeds may possess other advantages for sprint horses – for example, causing less gut-fill, or nonfunctional weight. Where unnecessary problems of 'hotting-up' have arisen, it is partly the consequence of a lack of appreciation of the differences between cereal grains in their energy content and bulk density described in Chapter 5 and quantified in Table 5.5. The cooking of cereal starch may reduce extended 'hotting-up' by promoting digestion and thereby decreasing microbial fermentation.

In contrast to *total* heat production, the *rate* of fermentation, and of heat evolved, from indigestible and 'spill-over' starch is more rapid than that from fibre, and an increase in metabolic rate is caused by the greater peak blood glucose level achieved by digestible cereal starch cf. roughages. This also increases the rate of heat production. This increase in metabolic heat production rate is the principal cause of the heat increment of food in humans and it is greater with foods yielding large glucose and insulin responses and an enhanced glucose storage as glycogen. These rates can be controlled to a considerable extent by the amounts of feed and the way this feed is presented (see Chapters 5 and 6). This elevated 'hotting-up' is of shorter duration than that of the total heat increment attributable to a feed and its effects should have passed before horses are subjected to strenuous effort, say five hours later, which would follow small feeds.

Gut fill and speed

Processing of roughage can accelerate rate of passage and reduce gut fill, but decrease hind-gut utilization (Wolter *et al.* 1975, 1977, 1978). The extent to which feed is fermented will influence the weight of ingesta. Slade (1987) measured the speed of Quarter Horses galloping at up to 19.6 m/s (44 mph) over 137 or 229 m from a running start. Speeds differed according to digestibility of feeds, as reflected by differences in gut fill.

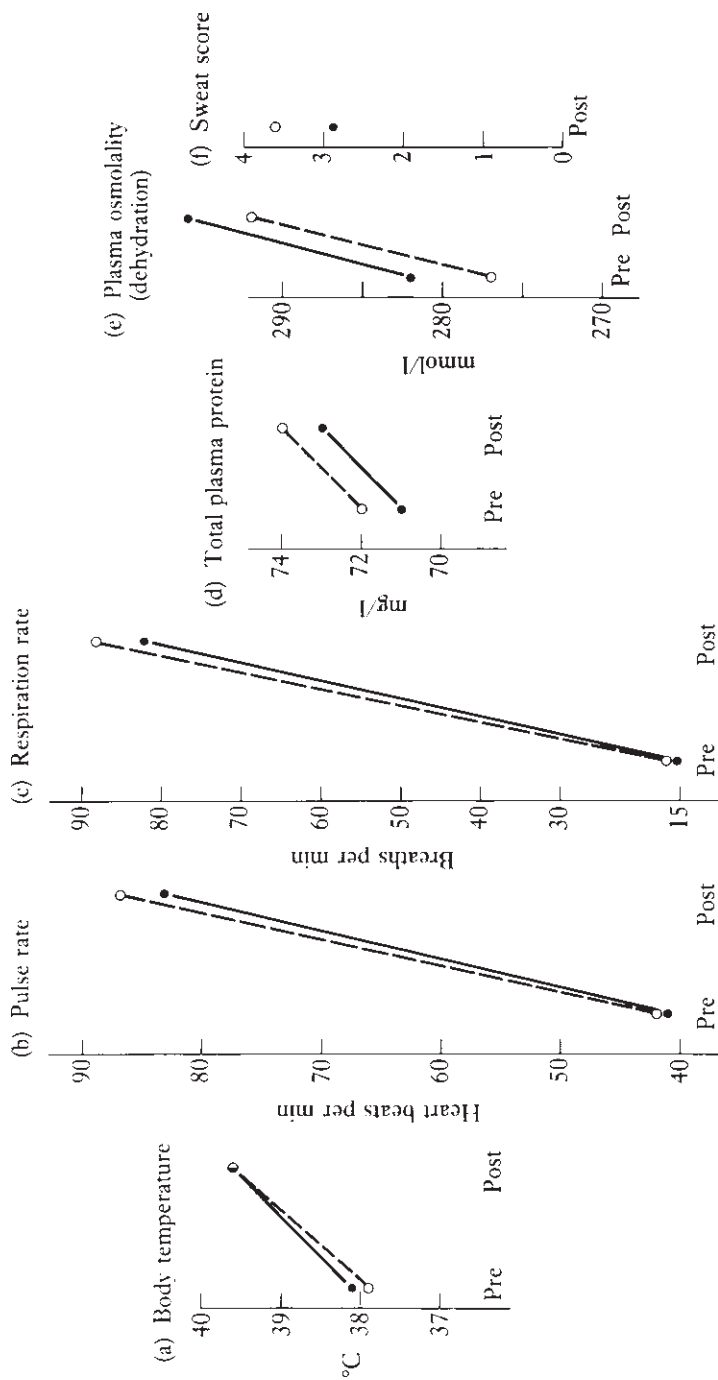


Fig. 9.19 Pre-exercise and post-exercise values for polo ponies given 6.8 kg lucerne hay plus 3.2 kg maize (●) or 8.2 kg timothy hay plus 4.0 kg oats (○) continuously over 4-week periods of a reversal experiment to maintain constant body weight (Wiltzie & Hintz 1983).

Feeding before endurance rides

Meyer (1987) concluded that endurance horses (cf. sprint horses) should be fed larger amounts of roughage (6–8 kg/day) to dilate the large intestinal volume, increasing water and electrolyte reserves. Poor-quality roughage should be avoided, as it causes a higher post-exercise plasma lactate concentration and more extreme hypoglycaemia than found with good-quality roughage. Warren *et al.* (2001) provided an afternoon feed, either high in fibre (54% NDF, 31% ADF) causing a 15% greater GI-tract volume, or low in fibre (31% NDF, 19% ADF). The morning feed was withheld and subsequent exercise with dehydration caused a greater loss of extracellular fluid with the high-fibre (HF) feed. However, the decline in plasma volume was similar for both, indicating to the authors that the extra GI-tract fluid with HF may have replaced circulatory losses more effectively.

The maximum postprandial increase in caecal volume is 8–14 kg/kg DM ingested, depending on the fibre content, with 130–135 mmol Na/l flow from the ileum. The average Na content of the large intestine is 40 mmol/l. Meyer (1987) concluded from previous evidence (Meyer *et al.* 1982a) that the horse should be fed at least five hours before an endurance race, depending on the feeding sequence (see 'Feed sequence, protein utilization and plasma amino acids'), as most of the residue will then have passed the ileocaecal orifice. Meyer (1987) compared two rations: one contained 2 kg concentrates and 3 kg hay, providing 11.5 g Na and 80 g K; the other contained 2 kg oats, providing 1 g Na and 10 g K. Four hours postprandial the retention of water, Na and K was, respectively, 5.8 kg, 9.3 g and 48 g for the first ration and 0.6 kg, 0.2 g and 1 g for the second. In support of Meyer, Ralston (1988) found that horses failing to complete 160 km races had received mixtures containing less hay and more grain, and had been trained for greater distances each week (83 cf. 61 km). The energy intake per kilometre trained was less in those that failed. Some samples of roughage contain excessive K which can stimulate diuresis, and so loss of water.

Glucose solutions given before prolonged exercise could increase glycogen degradation rate, through decreased fatty-acid mobilization, induced by the antilipolytic effect of elevated plasma insulin. Glucose given during hard exercise does not stimulate insulin secretion, but hyperinsulinaemia is protracted in TBs following a meal (Stull *et al.* 1987; Frape 1989), the timing of which may be critical before extended exercise. Ponies given a fluid providing 5.4 MJ DE following exercise daily exhibited lower heart rates and blood lactate concentrations during and following subsequent exercise (Lindner *et al.* 1991).

Feeding before sprints

In contrast, for short races there is some justification for a *light* concentrate ration four to five hours before the start, although timing is rather critical as the race should not coincide with elevated plasma insulin. A large meal would extend the time over which plasma insulin is elevated, and forage intake should be strictly limited. Rice *et al.* (2001) conditioned TB horses to 10.1 kg (*ad libitum*) or 4.3 kg

Table 9.13 Factors influencing optimum interval between last meal and subsequent exercise.

-
- Weight of ingesta
 - Size of meal
 - Rate of passage
 - proportion of roughage
 - degree of grinding of roughage
 - Dietary cation:anion balance
 - ≈250mEq/kg 3–4 h before sprint
 - Potential for yielding glucose or VFA
 - Digestibility of starch
 - insulinaemia
 - hind-gut volume and fluid/electrolyte retention
 - Fermentability of fibre
 - Hind-gut volume and fluid/electrolyte retention

Conclusions

- Type of meal: more roughage for aerobic exercise than for anaerobic.
 - Optimum interval: sprint 3–7 h, extended aerobic exercise 5–8 h.
-

(restricted) grass hay during the three days before intense exercise (feed and water were removed four hours before exercise), whereupon the restriction reduced body weight by 2%. Mass-specific $\dot{V}O_2$ was higher and both accumulated O_2 deficit and peak plasma lactate were lower in the restricted group, implying lower fatigue. The factors influencing the optimum interval between a meal and exercise are summarized in Table 9.13.

Meeting the increase in energy needs

The build-up of feed and of training before races may take 8–12 weeks, and in the case of event horses a typical build-up may start with 5 kg concentrated feed daily and finish with 8–8.5 kg two months later. The ration should be distributed among four daily feeds, and the hay, one-third of which should be given in the morning feed and two-thirds in the evening, should be reduced to 3.5–4 kg/day for a horse of average size during the last part of training. (The optimum sequence of concentrate and hay may be otherwise in the last days before an event, as indicated in ‘Feed sequence, protein utilization and plasma amino acids’, this chapter.) The protracted and intensive training for dressage (Plate 9.2) places emphasis on mental attitude and alertness, but it is equally important to achieve the right level of energy intake at each meal and overall. It is speculated that lecithin supplements may assist the mental composure of dressage horses, as may be indicated by the observations of Holland *et al.* (1996).

Feed allowances and work intensity

In all cases, feed intake should be increased as the work rate increases and the concentrated feeds should be severely restricted if work rate is reduced for any



Plate 9.2 Wily Trout, a 16-year-old gelding, ridden by Chris Bartle and Pinocchio, a 15-year-old, ridden by Jane Bartle in 'Passage' during dressage training for the 1984 Olympic Games.

reason, whether this be of short or longer duration. On rest days, a horse that would normally receive 8kg concentrates should then receive a maximum of 4kg distributed in three feeds, but with a greater allowance of hay of up to 5–5.5kg for the average-sized horse.

Either underfeeding or overfeeding leads to inferior performance. A horse should not be fed in order to fortify it with reserves for future events, but rather the rates of feeding should be consistent with immediate needs. Overfeeding will create fatness, which causes a greater burden on the heart and the horse generally, and it interferes with the dissipation of heat. The synthesis of fat from excess carbohydrate, protein and dietary fat does not encourage those enzymes that participate in the breakdown of fat so necessary during work. Furthermore, overfeeding can lead to stocking up (oedema) of the legs, hives (bumps under the skin), forms of colic, founder, exertion myopathy and general overheating.

Concentrated feed should be given at a minimum of three meals per day with some hay, ample water and salt supplements available, in the morning, at noon and with ample hay in the evening. Many horses in training do not take sufficient salt from licks, hampering progress, and a feed source providing 60g daily is recommended.

Feed intakes in practice

Observations both in the USA and the UK show that horses racing on the flat from two years of age, and weighing 470–530kg, consume daily 13–18.5kg total feed,

Table 9.14 Mean daily concentrate and roughage intakes by Thoroughbred (TB), Standardbred (SB) and French Trotter (FT) horses reported for commercial stables in France, Australia and the USA (Respondek *et al.* 2003).

	Total kg DM/day, per 100 kg BW	Concentrate kg DM/day	Hay kg DM/day
TB, 448 kg BW			
France, Respondek <i>et al.</i> (2003)	3.0	7.6	7.4
Australia, Southwood (1993a)	2.2	7.8	3.3
USA, Gallagher (1992)	2.4	5.6	6.6
SB or FT, 447 kg BW			
France, Respondek <i>et al.</i> (2003)	2.5	6.8	6.0
Australia, Southwood (1993a)	2.7	7.7	4.1
USA, Gallagher (1992)	3.2	5.1	9.3

which amounts to between 2.7 and 3.7% of body weight (Mullen *et al.* 1979; Hintz & Meakim 1981; Glade 1983a; D. Frape unpublished data). Of this, concentrates with, for example, cereals, nuts, bran and linseed, amount to 30–60% of the ration. In one American study of 171 horses (Glade 1983a) the concentrates provided 43–59% of the DE and 39–64% of the crude protein of the total ration. Moreover, the total ration provided 163 MJ DE/500 kg BW and 1686 g crude protein [129% of the NRC (1989) estimated minimum requirement]. In the UK, daily rates of protein intake among both flat and National Hunt horses amount to between 1000 and 1400 g/day (the author's measurements). These figures are well below the average of the American horses, largely as a result of the lower protein content of horse hays produced in the UK. Recently, Respondek *et al.* (2003) compared the feeding regimes of 29 French stables with the regimes published for similar stables in the USA and Australia (Table 9.14). Far less hay is offered in Australia than in the other two countries.

Appendix B gives examples of dietary compositional errors encountered by the author in practice.

Transportation

The method and extent of the transport of horses before events could be critical to their subsequent performance. There is, nevertheless, little published evidence on this. Van den Berg *et al.* (1998) showed that TB mares transported 600 km over eight hours in South Africa did not drink, but ate, during transportation and that potassium intake was decreased, so that water and electrolyte balances were affected. The stress of transport in mature horses over journeys of as little as two hours in France (Goachet *et al.* 2003) and of 24 hours in California (Stull *et al.* 2003) led variously to increases in WBC count, neutrophil:lymphocyte ratio, blood cortisol, heart rate, rectal temperature, faecal moisture content and weight of faeces produced. Thus, continued effort must be taken to reduce this stress.

STUDY QUESTIONS

- (1) How would you feed a horse from 96 hours before (a) a sprint event, (b) a major dressage event or (c) a long-distance event?
- (2) How should you manage a fatigued horse?
- (3) How would you introduce fat-supplement feeding to a stable of working horses?

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Chapter 10

Grassland and Pasture Management

... but that grass which grows on wet grounds, or the winter-grass, abounds with little or no spirit, wherein a great deal of the true nourishment consists, and therefore it must needs beget a viscid and indigested chyle, which must also render those horses that are fed with it sluggish, dull and unactive.

W. Gibson 1726

GRASSLAND TYPES

In humid temperate climates, natural succession favours the replacement of grassland by scrub and then woodland and forest. To sustain high-quality 'permanent' pasture requires perseverance in land management, through the grazing of domesticated animals and through the treatment of grassland as a crop to be cultivated. These pastures constitute the greater proportion of grazing and they contrast with the uncultivated areas of mountain, moorland, heath and downland, where wild grazing and browsing animals contribute to the distribution of plant species.

Fertility and grass species

The most fertile temperate pastures can theoretically support annually five, 500 kg barren or pregnant mares per hectare from grazing and conserved forage. The most productive swards contain more than 30% perennial rye grass (*Lolium perenne*), a proportion of rough meadow grass (*Poa trivialis*), and the remainder of grasses consisting mainly of cocksfoot (*Dactylus glomerata*), timothy (*Phleum pratense*), other meadow grasses, Yorkshire fog (*Holcus lanatus*), species of bent grass (*Agrostis*) and fescue (*Festuca*). The proportion of white clover (*Trifolium repens*) depends very much on the use of nitrogenous fertilizers and the seasonal grazing pattern, but can amount to 25% of the cover.

Other broad-leaved plants vary in abundance according to management. An extensive survey of grasslands in England and Wales (Hopkins 1986) (Table 10.1) indicated that *Lolium perenne*, *Agrostis* spp. and *Holcus lanatus* were numerically the most important species, contributing 35%, 21% and 10% of the cover, respectively (of those swards exceeding 20 years of age the proportions were 22%, 27% and 14%, respectively). The palatable *L. perenne* decreased in proportion with time and the unpalatable *H. lanatus* increased. The fescues (*F. arundinacea*, *F. rubra*) are also highly palatable to horses and generally their persistence requires a lower fertility than does perennial rye grass. In England, potential stocking density of all

Table 10.1 Factors favouring species distribution in pasture* (after Hopkins 1986).

	Drainage	Soil nutrient status	Fertilizer N inputs	Grazing intensity	Hay cutting	Sward age	Elevation	Optimum pH
<i>L. perenne</i>	Good	good	high	hard	—	young	low	6–7
<i>T. repens</i>	Moderate to good	good	low	hard	no	younger	—	7–7.5
<i>H. lanatus</i>	Poor	low	low	moderate	yes	older	NS	5–6
<i>F. rubra</i>	Moderate	low	low	moderate	—	older	high	5–6
<i>P. trivialis</i>	Moderate	high	high	low	yes	—	—	6–6.5
<i>Rumex</i> spp.	Poor to moderate	fair	—	—	yes	—	—	—
<i>Agrostis</i>	Moderate	low	low	low	—	older	NS	5–6

*It should be recognized that these assessments relate to mixed swards, as pastures of single species only exist as such immediately after sowing.

NS, no significant correlation between elevation and frequency of the species.

grazing animals is generally correlated positively with the contribution perennial rye grass makes to the sward.

Poor drainage, low fertility and plant species

Where poor drainage has not been rectified, creeping bent (*Agrostis stolonifera*), Yorkshire fog, rough meadow grass and creeping buttercup (*Ranunculus repens*) thrive better than ryegrass. Other less-productive grasses which invade swards of this class in significant numbers include meadow foxtail (*Alopecurus pratensis*), couch (*Agropyron repens*), crested dog's-tail (*Cynosurus cristatus*) and barley grass (*Hordeum murinum* and *H. pratense*), together with red clover (*Trifolium pratense*) and bird's-foot trefoils (*Lotus* spp.). However, the decline in ground cover by sown species over the years occurs on all but the most fertile land regardless of the excellence of drainage. About 20% of the cover by sown species is lost after 5–8 years and a further 10% is lost during the next 4–12 years. Poorly drained soils provide a less suitable initial habitat for sown species and the proportion of them is less throughout the pasture's life. Improvement by heavy treatment with fertilizers, drainage and intensive management yields swards of open texture, subject to poaching in wet weather. Lush pastures of this description, with little bottom, are unsuited for grazing by horses without great care and experience.

In many river valleys, rushes and sedges appear in *Agrostis* pastures where drainage is impeded, or the land is otherwise neglected. The fertility may be potentially quite high, but in the more degenerate and derelict land, even in lowland areas, purple moorgrass or flying bent (*Molinia caerulea*), bracken (*Pteridium aquilinum*) and gorse (*Ulex europaeus*), quite useless for horses, may sometimes appear. On better-drained slopes of acid soils, between altitudes of 100 and 350 m (350–1100 ft) under annual rainfalls of 90–120 cm (35–45 in), fine-leaved fescues and bent grasses

dominate pastures with a scarcity of clovers in the latitudes of 50–57°N of maritime climates.

The specific distribution within these ranges depends on soil pH, latitude, aspect, soil drainage and grazing. These areas merge into the uncultivated rough and hill grazings in which there is the invasion of bracken fern and gorse at lower altitudes along with the fine-leaved fescues. On moorland, matgrass (*Nardus stricta*) and purple moor grass, rushes (*Juncus* spp.), heather (*Calluna vulgaris*) and bell heather (*Erica cinerea*) may occupy a larger proportion of the area. Fertilizers will generally encourage nutritious grass species, whereas excessive grazing by ponies of the better fine-leaved fescue and *Agrostis* areas may lead to their suppression and the encroachment of economically useless *Nardus*, bracken, etc. In addition, where the pH is low, poor bone development occurs in young horses. The spread of shrubs and useless weeds may largely depend on drainage, the extent of cutting and the presence or absence of cattle.

Poor upland grassland is generally considered to be of marginal value for horse production. Nevertheless, in France, upland pastures composed of *Nardus stricta*, *Festuca ovina* and including *Vaccinium myrtillus* (bilberry, blaeberry) are grazed successfully in summer by heavy breed mares (1.5 ha per mare and foal), during which time the mares gain in condition (Micol & Martin-Rosset 1995).

Herb strips

Herbs may be defined as broad-leaved plants with nonwoody aerial parts and so could, of course, include clovers. Like clovers, many other herbs are rich in protein, minerals and trace elements relative to the common grasses; some are relished by horses and espoused by enthusiasts. The dry matter of nettle, for instance, contains nearly 6% of lime, 5% of potash and 2% of phosphoric acid, but the fresh plant is usually not sought by horses and ponies. A few relevant chemical values of herbs are given in Table 10.2. Herbs may be especially useful on marginal land, in which the upper layers are frequently leached of nutrients by excessive rainfall, and many tend to stay green in winter, so they provide a succulent winter bite although their regrowth is protracted.

When herbs are present in abundance they depress total yield per hectare of major nutrients, but this is less likely to be a critical issue in horse paddocks. In any case, their establishment in pasture as part of a normal grass and clover seed mixture is uncertain. Herb seeds are rather expensive, but many of them inevitably become established in permanent pastures through natural agencies. Herb strips are frequently sown on the headlands of fields. Table 10.3 gives suggested seed mixtures, which include some relatively noncompetitive grass species, although these are not essential. Chicory (*Cichorium intybus*) is a successful herb for strip-seeding in temperate pasture areas. It can reduce nutrient losses and yields DM rich in K, P, Ca, Mg and Na, although poor in N. However, the economic worth of herb mixtures is unproven for horses in any general way.

Table 10.2 Mineral contents (g/kg DM) of perennial rye grass and red clover at early maturity (Thomas *et al.* 1952; Worden *et al.* 1963) and of herb species (Hopkins *et al.* 1994) as means of four harvest dates from pasture sward.

	N	P	K	Ca	Na	Mg
Perennial rye grass						
Head	22.0	4.2	17.0	2.3	—	1.3
Leaf	21.0	3.2	23.0	8.7	—	1.7
Stem	8.0	2.7	17.0	3.0	—	0.9
Red clover						
Head	37.0	4.1	21.0	11.0	—	2.8
Leaf plus petioles	45.0	2.9	17.0	21.0	—	3.4
Stem	16.0	1.5	17.0	11.0	—	2.4
Chicory (<i>Cichorium intybus</i>)	26.2	5.4	26.1	19.0	12.2	3.5
Yarrow (<i>Achillea millefolium</i>)	26.4	5.8	33.2	12.1	13.0	2.5
Dandelion (<i>Taraxacum officinale</i>)	21.3	3.3	29.3	7.8	10.4	2.1
Ribwort plantain (<i>Plantago lanceolata</i>)	23.6	4.5	17.8	20.5	10.0	2.2
Grass/clover	24.2	3.5	19.3	9.9	3.9	1.6

Table 10.3 Suggested herb mixtures (kg/ha), including some grasses, for sowing as a strip 8–10 m wide in horse paddocks.

	Based on	
	Davies 1952	Archer 1978a*
Chicory (<i>Cichorium intybus</i>)	3	2.2
Ribwort plantain (<i>Plantago lanceolata</i>)	3	1.1
Burnet (<i>Sanguisorba minor</i>)	4	2.2
Yarrow (<i>Achillea millefolium</i>)	1	0.6
Cat's-ear (<i>Hypochoeris radicata</i>)	2	—
Dandelion (<i>Taraxacum officinale</i>)	—	0.3
Sheep's parsley, wild parsley (<i>Petroselinum crispum</i>)	1	0.6
Meadow fescue (<i>Festuca elatior</i>) or creeping red fescue (<i>F. rubra</i>)	3	(13)
Timothy (<i>Phleum pratense</i>) or smooth meadowgrass (<i>Poa pratensis</i>)	3	(7)
Crested dog's-tail (<i>Cynosurus cristatus</i>)	—	(7)
White clover (S100) (<i>Trifolium repens</i>)	3	—
Total	23	7 or 34

* Without the inclusion of grass seed the mixture should be introduced into an existing paddock by direct seeding if ground is well harrowed and the sward cut short.

Grass breeding

Institute of Grassland and Environmental Research (IGER), Traws goed, Aberystwyth, Dyfed, SY23 4LL, in Wales, has crossed early- with late-flowering perennial rye grasses to produce a more even DM production through the season. Rye grass $\times$ fescue (*Lolium multiflorum* $\times$ *Festuca gigantea*) hybrids have been bred to increase

pasture persistence and to resist drought. Italian rye grass containing 44% more Mg than standard Italian rye grass indicates it may be useful for hay production in studs. All these ventures could be of particular value in equine husbandry. Where grass is grown specifically for silage, a higher yield potential exists in some more exotic grass species, such as brome grasses. (For endophytes in seed production see 'Plant disease control', this chapter.)

PASTURE AS AN EXERCISE AREA

The production by pasture of digestible nutrients for horses and ponies is clearly of economic importance. Its critical role is starkly revealed by the historical evidence of the delay of European military campaigns until the spring flush in May, by armies dependent upon horses. A number of factors play a significant part in the selection of pastures, or their management, for horse husbandry. The thick cushion found in old pastures is better for exercise than is the open texture of heavily fertilized leys, in which numerous stones, upturned during ploughing, contribute to leg injuries.

Many horses needing rest and gentle exercise between periods of hard work, barren and pregnant mares and one-to-three-year-old growing stock are turned out to subsist on pasture. In each of these cases, leys or highly fertilized permanent pastures would initiate rapid and unwanted fat deposition. This creates unnecessary problems in the early stages of subsequent work, in late pregnancy and early lactation, or it contributes to leg abnormalities in growing horses and to the incidence of laminitis and colic. Therefore, a high degree of skill is needed in evolving pastures for horses and ponies that provide useful grazing and also yield saleable and reliable stock. The thick, matted turf of well-drained old pasture resists poaching in wet weather and can, therefore, provide exercise and maintenance areas for out-wintered stock. However, not only is the total annual yield of digestible feed generally lower in these pastures, but, especially where drainage is poor, the season of herbage growth is shorter, a fact probably of much greater economic significance. The length of the grazing season is generally greater the higher the fertility of the land.

NUTRITIONAL PRODUCTIVITY OF PASTURE

Within any pasture, the nutritional quality varies from area to area. Therefore, the feeding value of the whole pasture will depend on the stocking density and the amount of the most attractive herbage at any one time. In temperate grassland areas – excluding acid land with very high rainfall – the protein content of pasture is directly correlated with rainfall and inversely with soil temperature during the growth period. Pastures grazed by horses in northern temperate latitudes tend to produce the greatest yield of DE and protein during May and June, after which there is a precipitous decline in productivity from July to August when grasses

flower. Clovers and other legumes, if encouraged, prolong pasture growth and extend the summer grazing season.

Where persistent leafy strains of grasses have been established on fertile soils, this mid-summer fall in productivity is much less noticeable. More fertile deep soils are less inclined to dry out and the leafy strains of grasses continue vegetative growth much later into the summer. By grazing these pastures, the formation of seed heads is delayed, or avoided, and tillering encouraged, so that their productivity is further enhanced.

Regrowth of succulent leafy material occurs in early autumn, but work with sheep indicates that the ME of autumn grass is utilized 40% less efficiently than that of spring grass of the same crude chemical composition. This poorer value should be recognized when foals are weaned in the late summer on pasture without supplementary feeding (see Chapter 7).

Minerals

Excepting horses confined to tropical grasses, a Ca deficiency is unlikely among grazing horses, even when the grazing and browsing are desiccated. In a parched terrain, horses and ponies are deprived, first, of water (Table 7.4), energy and protein and, second, of P. However, stock can become deficient in Ca, P and Mg if they are confined to wet acid soils covered by poor-quality, fine-leaved grasses. Many ponies coming off such hill land present signs of 'big-head' and other consequences of bone demineralization. Horses seem to be less prone to grass tetany caused by Mg deficiency than are cattle, but a fall in serum Mg is possible when lactating mares are grazing on low-Mg soils and it has been suggested that part of the effect is through excessive amounts of K in lush herbage. Leafy material contains far more K than the horse requires in normal circumstances. The needs for Na and Cl are likely to be met in horses dependent upon pasture in temperate latitudes.

Vitamins

Green leafy material is a rich source of folic acid, and comparisons made by the author (unpublished observations) between horses in training for flat racing, given a cereal-based diet supplemented with folic acid and vitamin B₁₂, and grazing in-foal and barren mares, foals and yearlings indicated a 23% lower concentration of serum folate and a 33% lower concentration of serum vitamin B₁₂ in the horses in training. Several other water-soluble vitamins are equally adequate in cereal-based and grazing diets.

There are normally large stores of vitamin A in the liver resulting from the consumption of green herbage rich in β -carotene, but after a very extended drought there can be a clinical vitamin A deficiency as a result of protein and Zn deprivation coupled with the scarcity of green herbage. It is unlikely that a deficiency of any of the other fat-soluble vitamins D, E and K would occur among horses confined entirely to pasture. However, a few isolated pasture species not found in the UK

(see 'Vitamins D₂ and D₃', Chapter 4) can cause vitamin D toxicity, bone demineralization and soft-tissue calcification.

Trace elements

Australian evidence (Langlands & Cohen 1978) suggests that general pasture improvement increases the uptake by grazing animals of Cu, Zn, Mn, P, Ca and Mg. Improvement in the drainage of waterlogged soils tends to increase Se and Zn availability, but it may reduce the availability of Fe, Mn, Co and Mo, and an excessive use of N fertilizers may decrease the concentration of several trace elements in the sward. However, the relationships are complex (BurrIDGE *et al.* 1983).

The effect of drainage on Mo availability may be advantageous as peaty, poorly drained soils found in parts of Somerset and Ireland precipitate Cu-deficiency problems in ruminants through low availability of Cu and high availability of Mo in the soil, particularly where the soil pH is also high (see Chapter 3). These soils (pH in excess of 7.6–7.7) also tend to be deficient in available Mn and Co. Some of the soils contain more than 20 mg Mo/kg and an increase of Mo by 4 mg/kg depresses Cu availability to grazing ruminants by 50%. The effect may be seasonal and an excessive uptake by plants of Mo and sulphate in the absence of generous amounts of available Cu leads to deficiency signs in cattle and sheep. Hypocupraemia in horses occurs less widely, but it exists in several parts of the UK and particularly in Ireland. On the other hand, the horse is much less susceptible to the effects of Mo and sulphate, as in ruminants ruminal microorganisms synthesize thiomolybdate that reacts with Cu, decreasing its availability.

Soils subject to a high rainfall, waterlogging and a low soil pH are prone to Se-deficient herbage, as may occur in hill areas and on sands and gravels, in, for example, Newmarket, which is associated with low blood concentrations of Se in horses. By contrast, seleniferous soils containing very high levels of Se are a cause of toxic signs in grazing animals, for example on glacial lake deposits in Ireland. Shale, mudstone and clay soils contain higher concentrations of Se than chalk, limestone and sandstone soils (Thornton 1983) and many mountainous areas. Seleniferous soils are notorious in various regions of the world where accumulator plants store toxic amounts of soil Se. These accumulators leave Se residues which are apparently more readily absorbed by the roots of other plants, leading to alkali disease in grazing stock.

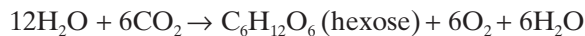
Some inland continental areas, and even alkaline soils in central England, can induce signs of I deficiency in the young stock of grazing mares. When seaweed is used in excessive quantities as a source, signs of I toxicity, similar to those of deficiency, have been observed (see Plate 3.1, p. 78). Deficiencies of Fe, Mn, Co and some other more exotic trace elements have not been recorded and are unlikely among grazing horses and ponies.

The correction of trace-element deficiencies by applying minerals to the soil is unsatisfactory for some elements as the uptake is scant and repeated treatment is necessary. Better absorption is generally achieved with foliar sprays, but these are

expensive and translocation is slight so that frequent treatment is unavoidable. Injections of Se have proved successful in grazing horses, but these are relatively expensive and repeated treatment at intervals is again necessary. When horses are held for extended periods on grazing lands, supplementary feeding with relatively concentrated sources of trace elements seems at present to be the most practical solution.

NUTRIENTS REQUIRED FOR PASTURE GROWTH AND DEVELOPMENT

Ultimately, nearly all life on this planet depends on sunlight and the fixing of atmospheric carbon by the action of chlorophyll present in bodies called chromoplasts in seaweeds and chloroplasts in higher plants (see also 'Photosensitization' this chapter) (some deep-sea bacteria derive energy from the oxidation of Fe). Chlorophyll is green, but the colour is masked by other pigments in some species. Chlorophyll is somewhat similar to haemoglobin, but contains Mg in place of Fe. It absorbs red, orange and blue parts of the spectrum and uses this radiant energy to combine water with carbon dioxide in a reduction reaction, producing hexose sugar and oxygen, summarized as:



It is clear that not only light and water are required, but the process also needs warmth, so pasture plant growth accelerates to a maximum in mid-summer, given adequate rainfall.

In addition to C, H and O, present as carbohydrates and fats, plant tissues contain a range of elements used in the synthetic process and present as components of proteins and many other tissue compounds. All these elements are present in many soils, but in most situations some are unavailable in optimum quantities for maximum plant growth, although in the height of summer water supply is frequently the limiting factor to this growth. The critical elements are:

- typically N, P and K, as provided in chemical fertilizers;
- the other major elements – Ca, S and Mg; and
- essential minor elements including Fe, Mn, Cu, Co, B, Mo, Zn, but also Na, Cl, Al and Si.

Leaching

Where fertilizers are applied to many soils, P and K are retained to a greater extent than is N. When N is not absorbed by plant roots, or used in microbial growth, much passes into drainage water. Concern exists over the pollution of streams, rivers and drinking-water sources with nitrates leached from the soil (Fig. 10.1). It has been estimated that only 8–16% of N entering pastures leaves farms in the UK as meat or

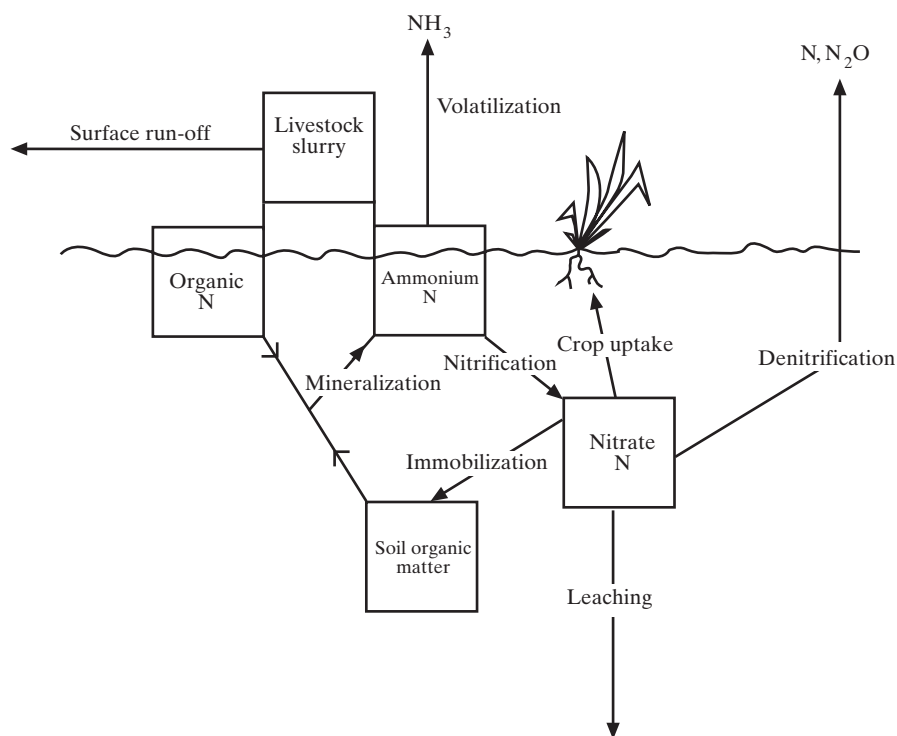


Fig. 10.1 Fate of slurry N in the soil. N can be lost as a result of surface run-off, volatilization of ammonia, denitrification and leaching (Anon 1986).

milk (rye-grass swards may recover 65–90% of N applied, but much is then recycled in dung and urine and ultimately lost) (Fig. 10.2). Apart from nitrates leached out, gaseous N is lost as ammonia (NH_3), nitrogen (N_2) and nitrous oxide (N_2O). Less N is lost when fertilizer is applied as ammonium sulphate than when it is applied as ammonium nitrate. Less still is lost when atmospheric N is fixed in root nodules of legumes by the bacterium *Rhizobium trifolii*.

Chemical composition of herbage and equine health

Amounts of crude protein, soluble sugars and nitrogen-free extractives (NFEs) in the dry matter of herbage are highest during the period of rapid leaf growth in the spring, next highest during regrowth in the early autumn, lower during the period of flowering in mid-summer and normally poorest during the winter when there is extensive dying back of the aerial parts of herbaceous plants. The months of the year when these phases occur in northern latitudes depend on the latitude, the lateness of the spring, rainfall, soil type and temperature. After the grazing of herbage, the first regrowth contains per unit of dry matter the highest protein, lowest crude fibre and highest NFEs, or soluble carbohydrate and starch. These values change progres-

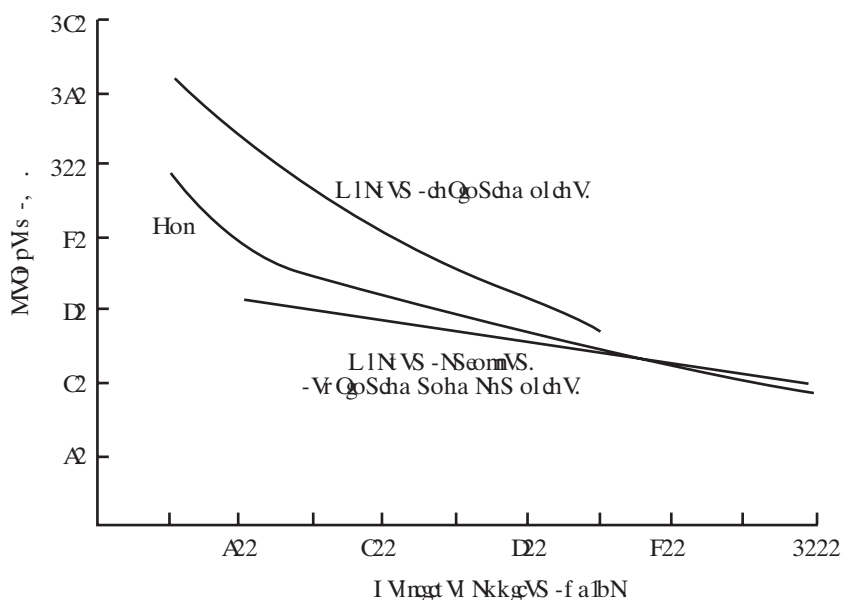


Fig. 10.2 Apparent recovery of N from cut and grazed swards in National Grassland Manuring trial GM24 during 1982–84 and apparent recovery from grazed swards when N inputs are adjusted for returns in dung and urine.

Table 10.4 Effect of growth of timothy (*Phleum pratense*) on the ratio of leaf to stem and chemical composition (% DM) (Waite & Sastry 1949).

Sampling date	Leaf/stem	Crude protein		Ether extract		Ash		Crude fibre		NFE	
		Leaf	Stem	Leaf	Stem	Leaf	Stem	Leaf	Stem	Leaf	Stem
20 May	2.57	21.7	14.1	3.8	2.9	7.1	9.9	19.1	23.5	48.3	49.6
2 June	1.30	17.2	11.4	4.7	2.5	6.5	7.9	23.8	29.7	47.8	48.5
16 June	0.39	18.5	7.6	4.1	2.6	8.0	6.6	26.1	32.6	43.3	50.6
30 June	0.35	12.3	4.4	3.3	1.7	8.8	5.0	26.9	31.7	48.7	57.2
14 July	0.20	11.1	3.4	3.2	1.3	9.0	5.0	30.6	32.4	46.1	57.9

sively as growth proceeds. For example, a study over 50 years ago (Fagan 1928) showed that in Italian rye grass (*Lolium multiflorum*) from the second to the tenth weeks of growth, the crude protein composition of the aerial parts declines from 19% to 7%, the crude fibre increases from 20% to 25% and the NFE increases from 44% to 60%. The changes can largely be explained by a rapid shift in the proportions of leaf to stem and leaf to flowering head (Tables 10.2 and 10.4).

The horse digests fibre less easily than can domesticated ruminants so that shorter grass containing a higher proportion of leaf is a more valuable feed than herbage approaching maturity. Temperate grasses contain large amounts of water-soluble carbohydrate (WSC) which consists of sucrose, fructose, glucose and fructans

Table 10.5 Mineral contents of grazed sward in stud paddocks at Newmarket, Suffolk (composition of DM) (data collected by author).

	Found	Normal range in UK
Calcium (%)	0.34–1.6	0.3–1
Phosphorus (%)	0.2–0.54	0.15–0.45
Potassium (%)	1.5–2.5	1.6–2.6
Magnesium (%)	0.12–0.2	0.11–0.27
Sodium (%)	0.03–0.34	0.1–0.6
Sulphur (%)	0.22–0.43	0.15–0.45
Molybdenum (mg/kg)	0.9–2.8	0.1–5
Copper (mg/kg)	4.5–12.3	2–15
Selenium (mg/kg)	0.025–0.049	0.02–0.15
Zinc (mg/kg)	21–34	12–40
Manganese (mg/kg)	44–220	30–115
Cobalt (mg/kg)	2.9–7.6	0.03–2

(oligo- and polyfructosyl sucrose). Fructans cannot be digested in the small intestine of mammals, but are rapidly utilized by microbial species in the large intestine. If large amounts of starch and/or fructans reach the hind-gut there is a rapid change in the microbial population, associated with the release of toxins and the onset of laminitis in susceptible horses. Moreover, cellulose fermentation *in vitro* with a pony faecal inoculum produces no lactate, whereas grass fermentation does, with high-fructan grass producing more lactate than low fructan (Longland & Murray 2003). It is, thus, important to establish the daily and seasonal variation in the fructan content of grasses. During one year in Wales, Longland *et al.* (1999) detected the highest content of fructans in *Lolium perenne*, var. Aurora and Perma leaf from May to September at midday and during early afternoon (24–42% of DM mid-afternoon, but a considerably lower proportion the following year), whereas the highest content in the stems occurred in the evening (see also Chapter 11, 'Laminitis and other diseases of the hoof'). Short lush grass is, therefore, more valuable only when it is used knowledgeably. Table 10.5 gives some mineral values for herbage found by the author in stud paddocks at Newmarket. Although these values change with stage of growth their digestibility is less affected by maturation than is that of energy.

White clover: its use and control and its relation to grazing intensity

Clovers tend to have deeper roots than do most grasses, especially the less-productive grass species. Clovers draw moisture and minerals from lower horizons in well-drained soils, so that they remain green during summer drought when grasses have gone to seed, they cause less N-pollution of drainage water and they may rectify an imbalance of trace elements between the upper and lower horizons of the soil profile.

Table 10.6 Effect of lime in seed bed with an initial pH of 4.7 and superphosphate in seed bed annually or in alternate years on annual DM yield (tonnes/ha) over 3 years* (Sheldrick *et al.* 1990).

Lime (t/ha)	Total* superphosphate application (kg P/ha)				
	0	3 × 15	2 × 30	3 × 30	1 × 120
3	5.19	6.22	5.54	7.35	7.50
6	6.82	7.38	7.09	7.45	6.38

* 15 kg annually, 30 kg in years 1 and 3, 30 kg annually, or 120 kg in seedbed.

Research interest has therefore refocused on making better use of white clover (or other legumes in nontemperate climates) in permanent pastures and leys. A white-clover sward can fix up to 200 kg N/ha annually, and use of this source can reduce N losses, mostly in winter, to a quarter of those resulting from equivalent fertilizer applications to a rye-grass sward. Although growth rate is slower where reliance is placed on clover N, this can benefit management of equine pastures.

Pastures on acid upland soils may carry no clover. White clover should then be sown as seed inoculated with *Rhizobia* of the appropriate strain, otherwise it will fail. In the UK, these strains are available from the IGER. Clover may also fail through lack of available phosphate, or excess ionic aluminium, caused by soil acidity. Liming at 3–5 tonnes/ha is therefore to be recommended (Table 10.6). Also slugs, weevils or eelworms (soil nematodes) may cause damage.

It is advised that the clover content of pasture be held in balance with that of grasses. Clover growth in the UK reaches a maximum in July, when it may represent the dominant weight of leaf in the pasture. The growing points of clover plants are farther above the soil surface than are those of grasses. Grass shoots, or branch stems, grow as 'tillers' (stolons on the surface) and as rhizomes (sub-surface) which originate at the nodes of the original stem, from which nodal leaves also originate. In perennial grasses, only a few of the stems are flowering stems, the remainder are vegetative stolons that in various grass species may be above ground, at ground level, or, in fact, are rhizomes below the soil surface. Grazing, especially by sheep, will remove relatively more leaf and growing points from clovers than from grasses. Therefore, repeated grazing can reduce the proportion of clover in the sward should this be required. In contrast, cutting for silage, liming and phosphate fertilizer application will tend to promote clover and 100–120 kg N/ha, applied per silage cut, will create a predominance of grasses.

White clover may be introduced by strip seeding with a Hunter rotary strip seeder at the rate of 4 kg Huia or Menna seed/ha in early summer, following paraquat spraying at 0.4 kg active ingredient (ai)/ha. The clover should be allowed to become well established before grazing and that should be on a rotational basis so that the cover is not reduced below 4.5 cm height.

Fertilizers, soil fertility and pH

A soil pH of approximately 6.5–6.8 should be maintained, as below this range there will not be enough free Ca and much of the P will be fixed as ferric phosphate or aluminium phosphate. Above the range, several minerals, including Fe, will be less available.

Phosphate fertilizer use

Clover, in particular, benefits from P fertilizer application, with the adjustment of soil pH if the soil is definitely acid. Basic slag, as a slow-release alkaline P source, applied every four to five years, is now a rarity, owing to changes in the steel industry. Superphosphate is composed mainly of $\text{Ca}(\text{H}_2\text{PO}_4)_2 + \text{CaSO}_4 \cdot 2\text{H}_2\text{O}$ (18% P_2O_5 , or 8% P) and is a readily available source of P not requiring to be as finely ground as the less-soluble slag and rock phosphate. It therefore has an immediate effect on P deficient pastures and is valuable for application to seedbeds. Where superphosphate and lime are shown to stimulate clover growth the soil is likely to have been acid and deficient in available P, and where pastures are rich in legumes then in all probability the Ca and P contents of the soil are satisfactory.

A visual survey of the frequency of clovers and scarcity of fine-leaved grasses can be used as a 'rule of thumb' in predicting the well-being of the soil in these respects. Triple superphosphate (45% P_2O_5 , or 20% P) in small amounts, i.e. 30 kg P/ha annually, or 120 kg/ha every four years (Table 10.6), with liming, should sustain clover presence on many mildly acid soils (pH 4.5–5.5). The amounts of pit chalk or limestone required on acid soils in order to raise the pH to within the desirable range and to ensure P availability will depend on the pH value and the texture of the soil, but amounts between 1.25 and 7.5 tonnes/ha can be used as a top dressing. In using limestone and rock phosphates it is critical that only finely ground material is purchased, as this characteristic will influence the availability of the Ca and P to the roots. Rock phosphates are not generally recommended for studs and are really suitable only on soils with a pH below 5. Limestone contains variable amounts of Mg and Mn that may be useful. Although limestone soils are rarely deficient in Mg, acid soils frequently are and dolomitic limestone would provide a useful source of this element.

Assessing fertility and fertilizer requirement

When first embarking on grassland management for horses and at intervals of, say, every ten years thereafter, it is desirable to carry out chemical determinations on the soil in order to assess, at the very least, its pH, P, K and Mg status. Soil sampling must be carried out in a representative and sensible fashion, even within a field, so that distinctions can be drawn between clearly different soil types. Furthermore, the soil profile in old pastures can be such that the status is quite dissimilar in upper and lower layers reached by plant roots. Many soils may show surface deficiencies of

Table 10.7 ADAS soil fertility scale (mg/l soil).

Index number	P	K	Mg
0	0–9	0–60	0–25
1	10–15	61–120	26–50
2	16–25	121–240	51–100
3	26–45	241–400	101–175
4	46–70	401–600	176–250
5	71–100	601–900	251–350

Table 10.8 Recommended rates of P, K and N treatment of grassland.

N, P or K index	For grazing (kg/ha) per year			For haymaking or silage – nutrients per cut (kg/ha)			
	P ₂ O ₅	K ₂ O	N ¹	P ₂ O ₅	K ₂ O	N High rainfall	Moderate ² rainfall
0	60	60	20–50	60	80	60	80
1	30	30	20–50	40	60	60	80
2	20	20	20–50	30	40	60	80
>2	0	0	20–50	30	30	60	80

¹The darker green the grass the less required. The larger quantities should be divided among three applications.

²45–65 cm (19–25 in) per year.

available P and Ca, accompanied by a lower pH, whereas lighter soils – especially where hay crops have been taken – are frequently K deficient in the upper layers. A full response to N fertilizers should not be anticipated if these primary deficiencies have not first been rectified.

Table 10.7 gives the indices used by the Agriculture Development and Advisory Service (ADAS) in the UK to classify grasslands in terms of their major nutrients. Where the value is 2 or over, no fertilizer treatment for that particular nutrient is required at the time of measurement. Recommended rates of P, K and N treatment of grassland for grazing and for haymaking or silage are given in Table 10.8.

Where fields are set aside for hay or silage a generous use of fertilizers is worthwhile. K fertilizer may be required only when a hay crop is taken, and it can be in the form of sulphate or ‘muriate of potash’ (potassium chloride), or as part of a compound fertilizer. Soluble Mg fertilizers are rarely used, but can be applied to increase the mobile Mg content of pasture herbage. Where K is liberally used this generally lowers the mobile Mg content of herbage. As horse pastures are frequently depleted of N, with yellowing of the grasses, the rates can be fairly high. On the other hand, the excessive application of N may cause laminitis and the pollution of drainage water. Even so, in order to maintain the quality of the herbage and to get an economic output from the pasture, moderate applications of N fertilizers should

Table 10.9 Approximate composition (%) of straight fertilizers and farmyard manure.

<i>Straight fertilizers</i>		
Ammonium nitrate	N	34
Ammonium sulphate	N	20.6
Nitrochalk	N	15.5
Calcium cyanamide	N	20.6
Triple superphosphate	P ₂ O ₅	45
Superphosphate	P ₂ O ₅	18
Steamed bone flour	P ₂ O ₅	29
Bone meal	P ₂ O ₅	22
Guano	P ₂ O ₅	13–27
Basic slag	P ₂ O ₅	18–20 (45–50 lime)
'Muriate of potash' (KCl)	K ₂ O	60
Sulphate of potash with magnesia	K ₂ O	26 (5–6 Mg)
Kainit (Na, K, Mg)	K ₂ O	14
Kieserite (MgSO ₄)	Mg	16
Calcined magnesite	Mg	60
<i>Farmyard manure</i> (kg/10t)		
	N	15
	P ₂ O ₅	20
	K ₂ O	40
	Mg	8

always be considered desirable unless there is a reasonable proportion of well-distributed clover. A sward content of 25–35% of red or white clover may yield the equivalent of 150–200 kg slowly released N/ha annually through N fixation. N application rates of 20–25 kg/ha, when required, three to six weeks before the pasture is grazed are recommended.

The composition of some straight fertilizers is given in Table 10.9. Compound fertilizers contain two or more nutrients in a reliable form and the weight of a nutrient in a 50 kg bag of any fertilizer is given by dividing the percentage of the nutrient by 2. Thus, a 50 kg bag of an N:P:K compound 20:10:10 will contain 10 kg N and 5 kg each of P₂O₅ and K₂O. In all situations, granulated, rather than powdered, inorganic fertilizers should be used and at least a week should elapse after treatment before horses and ponies are allowed onto established pastures. This will give sufficient time for the granules to percolate down to soil level, avoiding the consumption of any significant amounts by grazing animals.

The principal organic fertilizer is farmyard manure, preferably excluding horse manure, and its very approximate composition is given in Table 10.9. Its advantage over inorganic N sources is the slow release of N, but set against this is the expense of transport and distribution. It is probably more practical to distribute 50 tonnes/ha on a few paddocks than to use half as much on a much larger area, and there is some justification, particularly on light soils, for applications every five to seven years. Fish-meal, now scarce, containing 10–11% N, is sometimes used in organic-based fertilizers. It has advantages, in terms of slow release, similar to those of farmyard manure.

Table 10.10 Recommended rates of nutrient application to seedbeds in establishing a pasture (kg/ha)

P or K index	N					P ₂ O ₅ (All mixtures)	K ₂ O	
	Grass Spring sown	Autumn sown	Grass/ clover		Grass/ lucerne			Lucerne
			(a)	(b)				
0	125	50	40	75	50	25	100	125
1	100	50	0	50	25	0	75	75
2	75	0	0	0	0	0	50	30
>2	0	0	0	0	0	0	25	30

(a) If autumn sown or if it is intended to rely on clover as the main source of N for the sward.

(b) If little reliance is to be placed on clover as a source of N for the sward.

Where land has been ploughed up and new permanent pastures, or leys, are being sown, readily available nutrients should be provided for the seedlings; recommended rates of seedbed application are given in Table 10.10.

SWARD HEIGHT

A considerable number of studies have investigated the effects of the intensity of grazing, or cutting, on the productivity of pastures. Horses and sheep graze more closely than do cattle. Rye-grass and white-clover swards maintained at an average height of 3.6–3.8cm with sheep have been shown to produce more DM annually than those maintained at 2.5cm. With grazing cattle, similar unfertilized sward heights of 6cm were shown at IGER to be more productive and to retain a greater presence of clover than swards maintained at 4.5cm. The DM yields differed by 17% and 15% and clover presence differed by 4% and 23% in years one and two, respectively.

Reference was made to tillering (see ‘White clover: its use and control and its relation to grazing intensity’, above) and therefore the spreading of grasses in a sward, as influenced by grazing. In contrast to the effect on clovers, grasses are, within limits, encouraged by grazing if adequate leaf remains, and consequently they produce thousands more tillers per square metre of sward (Table 10.11). (Clovers also spread by producing thousands of stolons per square metre, but grazing causes a proportionately greater loss of leaf than in grasses.)

It is clear (1) that repeated cutting for silage/hay leads to pastures that are less suitable for horse grazing, as there is a less firm mat of herbage for exercise, (2) that close cutting in spring reduces subsequent grass seed production, and (3) that repeated close grazing reduces the proportion of clover in the sward and the total annual yield of DM.

Table 10.11 Effect of maintained sward height on tillering and proportion of tillers that produce flowering heads mid-June in a Melle perennial rye-grass pasture in the UK (Treacher *et al.* 1986).

	Cutting date		Grazing height (cm)		
	Mid-April	Late summer	3	5	7
Tillers (1000 s/m ²)	24	11	35	29	24
Flowering tillers (%)			20	27	47

INTENSITY OF STOCKING WITH HORSES AND RUMINANTS

Horses should be removed from a pasture as soon as they have eaten the available herbage, if alternative land exists. They are more active than ruminants, and they can damage both the soil's structure in wet weather and the growing plants through prolonged trampling. In contrast to cattle and sheep, which spend periods ruminating, horses may spend up to 60–70% of the day searching for the most delectable foliage. Overgrazing, poaching of the soil or stocking grassland during a heavy frost damages plants and depresses the rate of regrowth, encouraging the spread of prostrate and opportunist annual weeds seen particularly in an arc around gateways, drinkers and feed troughs. One of the few advantages of old, matted, sod-bound pastures is that they may be less prone to damage in this way. However, the undergrazing of pastures tends to promote the nutritionally poorer grass species, partly through seeding, and parasite survival, as distinct from transmission, is extended.

The ideal stocking rate is no greater than that which will feed the horses in the growing season. It is preferable to stock few horses with the balance made up of cattle to clear the excess growth at the season's height. Mixed stocking, either on a rotational basis or together with horses, initially decreases the number of horses that can be maintained, but these few will receive a better diet and will ingest fewer intestinal parasitic worm larvae. Moreover, the quality of the pasture can be maintained at a high level for many more years. By breaking up an area of land into paddocks, rotational grazing is facilitated and better parasite control is achieved, particularly where the grazing species are also rotated in each paddock. Occasionally, lactating mares or young stallions will bully cattle, so careful judgement should be exercised and rotation, rather than mixing of animal species, thus carries certain advantages.

Herbage yield and horse productivity

How does horse grazing affect pasture productivity and animal productivity? What degree of defoliation is optimum for horse productivity? Close cutting, or grazing,

reduces yield, but as herbage leaves mature and die they obstruct light to the newly emerging, actively growing leaves. Moreover, the digestibility of the pasture crop changes radically with stage of growth, and differs between stem and leaf. Maximum rate of leaf growth and yield of digestible nutrients for horses over the year require that leaf is harvested at some intermediate stage and that dead herbage is removed.

Sound pasture management becomes increasingly important as the area available to each horse is reduced, when conditions become increasingly favourable to parasite transmission and bullying, and when excessive stocking depresses herbage regrowth (through removal of leaf and clover growing points; see 'White clover: its use and control and its relation to grazing intensity', this chapter). In the New Jersey summer, total grass density (number of tillers/m²) was greatest with the highest stocking density, owing to the increase in density of tall fescue (*Festuca elatior*) at the highest stocking rate. The densities of all other species declined in summer compared with the spring (Singer 2001, personal communication). Singer considered that their optimum stocking density was 0.6–0.8 hectares per mature horse when Kentucky bluegrass (*Poa pratensis*) and annual bluegrass (*Poa annua*) represented 76% of the total grass density and common white clover (*Trifolium repens*) was the dominant legume.

There is a limit to the amount of trampling that grass can survive, particularly when soil is wet. The resulting bare ground becomes infested with docks, nettles and thistles. Thus, satisfactory husbandry is based upon:

- grazing paddocks just before grass-leaf growth declines, but well before senescence supervenes;
- allowing time for the grass to recover;
- replacing depleted nutrients;
- determining optimum interval between grazings (which varies with season, rainfall, ambient temperature and soil fertility);
- applying an understanding of the life cycles of the critical pasture worm parasites of horses (Chapter 11);
- understanding the temperament of each horse; and
- applying an understanding of each horse's environmental needs.

The horse digests forage less efficiently than do cattle. It therefore prefers, and obtains greater sustenance from, the younger leaves, but ample good pasture on its own can provide sufficient energy and protein for growth of horses over five to six months of age. Where the horse is forced by circumstances to graze less digestible parts of the herbage, some evidence indicates that it compensates by increasing its intake. This observation is not acceptable generally. Experiments in the Netherlands (Smolders & Houbiers, personal communication) showed that grass cut at the haymaking stage, yielding 3700 kg DM/ha, was consumed daily by adult horses at the rate of 2.1 compared with 2.4 kg DM/100 kg BW of grass cut at the grazing stage, yielding 1900 kg DM/ha (these are equivalent to 100 and 113 g/kg metabolic weight, respectively). The younger grass had higher energy and protein contents and a lower crude fibre content. An optimum was, however, found. The DM consumption rate

of grass with a DM content below 14% was less than that of grass above 14% DM. Thus, apart from extremely young grass, there is a decrease in grass DM intake with increasing maturity of that grass. These Dutch horses obtained, as a percentage of their maintenance requirements, 170–200% energy and 460–500% digestible protein. Grace *et al.* (1998a) reported that TB filly yearlings gained 0.75 kg/day on a New Zealand pasture containing 201 g crude protein/kg DM and 11.4 MJDE/kg DM. The DM intake was 6.5–7.5 kg/day (approximately 2 kg DM/100 kg BW) yielding an adequate daily DE intake of 81 MJDE.

Any guidelines on pasture use must be very approximate as all pastures differ in productivity. The husbandry of growing horses has the objective of producing not only efficient weight gain, but also sound skeletal growth (see Chapter 8). This twin objective has a bearing on recommendations for stocking density. Aiken *et al.* (1989) measured the weight gain of Quarter Horse yearlings of 347 kg BW at stocking rates of 6.7–12.4/ha Bermuda-grass (*Cynodon dactylon*) pasture. Weight gain per horse decreased in a complex way with increasing stocking rate. This probably related to the effect of grazing intensity on pasture yield of highly digestible herbage. Forage samples were vertically divided into top-, middle- and bottom-canopy layer thirds for analysis. The top third was that over 8 cm. Forage availability decreased in each of the three canopy layers as grazing pressure increased, but particularly in the least-dense, palatable top third. This third is the most photosynthetically active leaf tissue, the most productive both for horse growth and, of course, for pasture growth. With increased stocking rates, grazing of the lower two thirds became unavoidable. As these layers contain stems and senescent plant tissue, animal performance was limited by the small proportion of leaf present. The data are summarized in Tables 10.12 and 10.13.

Stock-carrying capacity

Grazing pressures on pastures with continuous grazing should be set low enough to ensure sufficient regrowth of vegetation, but high enough to prevent accumulation of mature, poorly digested forage. As horses select the tender, top layers of forage, this situation argues for complementary use of ruminants which clear and utilize mature and senescent plant tissue.

Table 10.12 Productivity of Bermuda grass pastures over 56 days grazed by yearling Quarter Horses (Aiken *et al.* 1989).

Yearlings/ ha	Herbage mass (kg DM/ha over 56 days)	Herbage allowance (kg DM/100 kg BW)	<i>In vitro</i> digestible DM (g/kg DM)	Weight gain per horse (kg/day)	Withers height increase (cm/56 days)
6.7	334	69	391	0.37	2.2
8.0	130	32	406	0.13	1.6
9.5	93	27	409	0.31	4.2
12.4	54	20	394	–0.31	2.0

Table 10.13 Productivity of Bermuda grass pastures in the top, middle and bottom layers of the canopy over 56 days when grazed by yearling Quarter Horses (Aiken *et al.* 1989).

Yearlings/ ha	Forage availability (kg DM/100kg BW)			Digestible DM* (g/kg DM)		
	Top	Middle	Bottom	Top	Middle	Bottom
6.7	10	21	38	421	403	383
8.0	4	11	17	442	425	404
9.5	2	11	14	413	405	392
12.4	1	8	11	414	400	391

*Measured *in vitro* by the method of Goering & Van Soest (1975).

When an allowance is made for loss of much of the edible foliage as a source of horse feed, but ignoring extremes of climatic fluctuations, the carrying capacities have been estimated: 1 ha high-quality grassland should provide pasture and hay for three to four light horses of about 400 kg, or four to six smaller ponies. Low-quality permanent pasture, however, may support only one horse per hectare and, in the extreme, 25 ha dry range may be required to supply the needs of a single horse throughout the year. Average-quality grassland can produce sufficient growth for two horses, and, with adequate moisture, when fertilized, for three horses per hectare, or, as summer pasture only, for double the number. On good temperate summer grassland in France, heavy-breed mares (700–800 kg BW) require 0.7–1.0 ha per mare and foal, or 1.5–2.0 ha when following cattle. With a rotational grazing system, the cutting of surplus herbage and N fertilization (80–150 kg N/ha), 2.0–2.5 growing horses are grazed per hectare. In contrast under harsh upland conditions in France only 0.5–0.7 growing horses per hectare are possible (Micol & Martin-Rosset 1995). Many TB studs produce barely enough grass for one mare plus followers up to yearlings sales per 1–1.5 ha and then provide none of their own hay. The latter should not in any event be produced from paddocks that have been grazed by horses during the previous year at the least, if worm control is to be practised assiduously.

Archer (1978a) found that only 10% of the area of long-established horse pastures was grazed. After ploughing and reseeded following an arable rotation, the grazing area was extended to 20–30%, most of which included the previously grazed areas. Horses will not graze near horse droppings and these areas are rejected if removal of the droppings is delayed for more than 25 hours after they have been voided. Horse urine does not engender a similar instinctive reaction. The inborn habit reduces the transmission of parasitic worm larvae and leads to the establishment of both grazing and camping areas, with a consequent effect on productivity. Horses will, however, graze right up to cattle dung pats and graze evenly over areas after well-rotted cattle manure has been spread. Similarly, cattle will graze the longer grass around the horse dung pats. Ideally, therefore, horse droppings should be removed from pastures on a daily basis and not spread by harrowing. Although

the latter practice will destroy more parasitic worms, it will enlarge the rejected area. The advantages of integrating ruminants with horse-pasture management is obvious. Steers are better than milking cows as they remove less Ca, P and N from the soil and both are probably better than sheep for which, nevertheless, there are some staunch advocates. Pastures heavily fertilized for dairy cows are generally unsuitable for horses.

Even with the best will and resolute adoption of mixed grazing, some areas of rank growth of low feeding value will remain, swamping out any young basal growth. Such areas should be topped at least six times per season, with the toppings removed to avoid mounds of mouldy grass suppressing the underlying grass. The pursuance of this practice may prevent weeds from seeding, it will promote tillering and regrowth of young grass and will destroy some of the infective larvae.

All grazing animals thrive better if they have the companionship of other stock and this may be particularly true with highly strung hotblooded horses. Even goats, sheep, chickens, ducks or ponies can fulfil a useful role. The old adage 'to get his goat' implied that a favourite racehorse could be nobbled by stealing his mascot before a race. Gilbert White, in his *Natural History and Antiquities of Selborne* (1789), relates in a letter dated 15 August 1775 to Daines Barrington how a lone horse made an abiding companion of a solitary domestic hen, consoling and protecting it, in so far as it was possible, from the trials of avian life.

In addition to companionship, all horses should have access to shelter from the sun at high noon and from cold, windy wet weather. This shelter may be naturally formed by trees or be a simple three-sided covered structure.

Booth *et al.* (1998) assessed the effects of wetting on heat loss of mature Shetland pony stallions in winter coat. The ponies were wetted on the back with water 5.26°C, housed at an ambient temperature of 2.0–9.5°C in an open-sided shed and given meadow hay at maintenance levels. Heat production (HP) was measured by indirect calorimetry (see Chapter 6):

$$\text{HP (W/kg)} = \{(\dot{V}\text{O}_2 \times 270.5) + (\dot{V}\text{CO}_2 \times 82.7)\} / \text{live weight (kg)} \quad (\text{Brouwer 1965})$$

Where W = watts, $\dot{V}$ = volume at standard temperature and pressure

Skin temperature decreased, but not rectal temperature and HP was not increased over three hours. The response would also depend on wind velocity and on the nature of the shelter. In my own experience, acclimatised and adequately fed horses are perfectly safe at –40°C. At this temperature the coat is not wet!

Extending the grazing season and rejuvenation of pasture

A continuous supply of inorganic nutrients, including water, is required for the growth of pasture plants. In addition, roots require oxygen, so soil moisture must be present without waterlogging. For this reason, the structure, type and humus content of soil are critical in ensuring a continuous supply of moisture at all levels through which the roots of pasture plants permeate. Cultivation to assist aeration

and to extend the grazing season is brought about by relieving compaction and poaching of well-established pastures. Earthworms play an important role in soil aeration and cultivation is of most benefit in wet areas. Paraplowing, moling or subsoiling all improve water-table levels and increase early season yields.

It is not only economically important to extend the grazing season, but also ecologically desirable to reduce leaching of N from the soil, which occurs outside the normal season of growth. Mixed pasture species, including improved grass strains, assist in the achievement of this goal. Rye (*Secale cereale*), strip-seeded in autumn into permanent pasture, increases early-season growth and it may be useful on light-textured free-draining soils in dry areas where N fertilizer use is restricted.

GRAZING BEHAVIOUR

There is a view that some environmental factors contribute to bad behaviour, even cribbing, among horses at pasture. What can be considered a poor-quality environment as seen through a horse's eyes, is learnt by experience. The proximity of other animals as company, ample palatable forage and some natural shelter from extreme conditions probably stand high in the estimation of most horses.

A crude comparison with the ruminant indicates that the horse has a much smaller stomach, necessitating short grazing sessions at relatively frequent intervals throughout the day. A study with mares allowed to graze during 12 hours light and 12 hours dark in North Carolina indicated that grazing took up 17.2 hours daily, in which 89.7% of the daylight and 76.4% of the dark were occupied in the activity. Horses tend to graze at similar times, but generally without interference once a hierarchy has been established. Dominance hierarchy or patterns of agonistic and affiliative behaviour are apparent during feeding from a single source, when horses are in close proximity to each other.

The rate of intake of forage during grazing depends on the quality of herbage, its density and the appetite and size of the horse. Work by Cross *et al.* (1995) confirms that grazing endophyte-infected (*Acremonium coenophialum*) tall fescue (*Festuca elatior*) (see 'Poisonous plants', this chapter) depresses grass DM intake and body weight maintenance of horses. On the other hand, healthy tall fescue and creeping red fescue (*Festuca rubra*) are two of the most palatable grass species for horses.

Experiments in Kentucky (Cantillon & Jackson, personal communication) with Quarter Horse geldings showed that the consumption of Johnson tall fescue (*F. arundinacea*) or lucerne (*Medicago sativa*) over seven hours of grazing averaged 5.5 kg organic DM, which was adequate for maintenance. There were 16–20 bites/min for grass species and 8 bites/min for alfalfa, so that each bite took in 0.7 g organic DM grass, or 1.7 g organic DM alfalfa. However, Naujeck & Hill (2003) demonstrated that, at least with swards of *Lolium perenne*, of leaf heights of 3–19 cm, bite size was roughly proportional to sward height in which the horse removed 51–68%

of the grass length at each bite. Bite rate per minute was not reported, so whether or not consumption rate was proportional to height is not established.

Soil ingestion

The ingestion of soil while grazing can occur in significant amounts, depending on the height of the herbage, the openness of the sward, the contamination of leaf by earth and on the species of animal. On rough terrain the soil intake by sheep is said to approach 20% of their daily intake of DM and measurements have shown that a 500 kg horse may ingest as much as 1–2 kg soil daily while grazing. Apart from K, the mineral and trace-element contents of soil are generally higher than they are in herbage DM, although their availability, or digestibility, varies with the element and soil type. Most common mineral elements (possibly apart from I and Co) are required by growing plants. However, the proportions taken up by roots differ considerably from one element to another. Plants do not absorb heavy metal poisons such as Pb [cadmium (Cd) may be absorbed in greater quantities] in significant quantities, although the leaves can become contaminated by industrial fallout and the soil and subsoil can be polluted by industrial seepage. Soil consumption may then be a cause of, for example, Pb or F toxicity. In the author's experience, lead shotgun pellets admixed with grass silage destroyed a high-yielding dairy herd. However, high concentrations of toxic minerals in the soil may solely depend on their geological origin. The significance of seleniferous soils is discussed elsewhere in this chapter (see 'Trace elements', 'Toxic legumes and other species' and Table 10.20; and also Chapter 3).

SUPPLEMENTS ON PASTURE

The nutrient value of pasture varies particularly with:

- season;
- climate;
- plant species;
- soil type;
- fertilizers; and
- geological formation.

The prime effect of soil type is on the trace-element and mineral content of the plant. The Ca content can be influenced to some extent by liming, and trace elements by foliar sprays. Possibly the most economical way of overcoming pasture deficiencies of trace elements in particular, is by directly supplementing the horses. Supplementation carries with it the risk of harmful excesses of certain of the trace elements, unless horses are dosed individually. For many years, mineralized salt licks have been available. These licks are unsatisfactory as they can be washed away

Table 10.14 Declared composition of molassed feed block used by Murray (1993).

Ca (g/kg)	90
P (g/kg)	50
Mg (g/kg)	40
Na (g/kg)	80
Cu (g/kg)	5
Zn (g/kg)	4
I (g/kg)	0.1
Se (g/kg)	0.015
Mn (g/kg)	1.5
Co (g/kg)	0.1
Vitamin A (iu/kg)	333 000
Vitamin D ₃ (iu/kg)	66 000
Vitamin E (iu/kg)	2 500

and some individuals ‘hog’ them, whilst others neglect them. Feed blocks are another form of supplementation.

Feed blocks

Hardened and molassed mineral feed blocks containing all the important major and minor mineral elements on a cereal base have now been available for many years (Table 10.14). Blocks are hardened by pressure and binding agents, or by the use of chemical setting reactions. These reactions include the formation of calcium sucrosate (molasses combines with calcium oxide or hydroxide, but the product is said to restrict intake, owing to a bitter taste), or calcium sulphate (gypsum). The author’s own experience with these blocks is that those that are well formed withstand quite wet weather, when they are held in appropriate containers, and that most horses at pasture feed from them. Nevertheless, there is considerable individual variability in consumption with time and age of block. Feed blocks should be protected from contamination in a container, but that container should allow consumption by prehension as well as by licking – the horse should be able to bite the edges. Murray (1993) placed blocks in cut-down plastic barrels, stabilized in the lower half with concrete. The barrels measured approximately 0.7 m high by 0.4 m wide. The height had the advantage of allowing sheep to graze with the horses without the risk of Cu intoxication.

A detailed examination of the consumption of molassed feed blocks was undertaken by Murray (1993) in Ireland. The following were the main conclusions:

- Weather in Ireland did not influence intake.
- Weight of block remaining influenced intake. Below a residual weight of 4 kg, intake decreased markedly.
- Intakes of Ca, Mg and P from herbage and the block together were higher than that recommended by the NRC (1989), and the calculated intakes of the various

trace elements were at all times below the maximum tolerance levels given by the NRC (1989).

- Plasma Ca and Mg concentrations did not reflect supplementation, whereas plasma and hair Cu concentrations increased with supplementation.
- Peak attendances at the block occurred early in the morning and at midday; the horse spending the longest there ate the most. Dominance hierarchy within a group influenced access to the block and consequently affected intake.
- Daily intake per horse ranged from 23–283 g, but averaged over a week the range was 32–215 g per day.

SAFETY OF GRAZING AREAS

Wooden fencing

The paddock principle of rotation allows a more complete recovery of the grass and prevents the excessive spread of flat weeds, such as plantain, daisy, buttercup and dandelion. All horse paddocks, structures and fences must be free from protruding nails, barbed wire, loose wire, tins, broken bottles, large stones and anything that can ensnare horses, particularly where young stock are reared, as they tend to be more curious and flighty.

Creosote is now considered to be too risky for the treatment of fence posts, and good substitutes are available. These are effective when applied properly, normally allowing the posts to soak thoroughly. Any wood treatment is best carried out in the open, to avoid inhaling any fumes. The posts should be allowed to dry thoroughly before horses have access to them. Horses should have no access to containers of tar or other chemicals containing phenolic substances, as phenol is very poisonous to horses, and is rapidly absorbed through intact skin. Wooden railway sleepers cut lengthways make good posts. Wooden fencing is readily protected from chewing by running electric fencing wire along the top (Plate 10.1). The tops of fence posts should be flat, with no sharp edges and level with the horizontal poles nailed to their inner sides.

Electric fencing

Stud managers have been reluctant to use electric fencing for controlling grazing. However, research has shown that single-strand reflective tape is effective, as long as the current is maintained continuously. One study in North Carolina with TBs, Quarter Horses and yearlings showed that horses, in groups of two or four, could be successfully rotated through 0.06 ha sections, back grazing the previous section. Occasional breakages of the fence occurred. The area was surrounded by three-wire electric fencing.



Plate 10.1 Use of electric fencing installed on post-and-rail fencing to discourage wood chewing. However, the protruding posts and nails, especially on the top rail, present a hazard to horses. Evidence indicates that electric wire attached directly to the wood provides an adequate deterrent, as wood is a sufficient insulator.

WATER SUPPLIES

A piped water supply should be clean and adequate (see Table 7.3, p. 255) and, where natural watercourses are used, freedom from contamination for at least a few miles upstream should be confirmed (see Chapter 4 for effects of drought on the safety of natural water sources).

SILAGE AND HAYLAGE AND THEIR SAFETY

(See also Chapter 5.) Silage quality is normally determined by the date or time of cutting. As grass grows during the spring and early summer yield increases but digestibility falls. In terms of the ruminant D-value a decrease of 1 unit of D-value between 71 and 51 is equivalent to an increase in dry-matter yield of perennial rye grass of 500 kg/ha, through delay in harvesting from the end of April to mid-June in the UK. The quality of forage legumes depends on the retention of leaf during harvesting. This is particularly true for lucerne, owing to the relatively high proportion of fibrous stem.

Silage should be made with safe raw materials free from weeds and contaminating pollutants. It should have undergone an adequate acetic acid fermentation and possess a high dry-matter content without any evidence of moulding, which can occur in the spaces at the tops of silos where the conditions are hot and humid. Good-quality haylage is quite satisfactory and material compressed in plastic sacks with a mild fermentation and with about 50% of dry matter is normally reasonably safe, but is usually on the expensive side. To prepare haylage, stands of pure grass are cut at the bud stage and wilted for 18–24 hours before baling tightly. Careful harvesting should minimize contamination of the herbage with earth. The baled product has a relatively high pH (about 5.5) and should not be fed to horses for at least a week after baling. Haylage from an unfamiliar source should be given in small quantities initially, even where the stock have previously received silage or haylage. Owing to its moisture content, its feeding value is about half that of high-quality nuts. However, its high content of soluble dry matter and ease of mastication facilitate a rapid intake of available energy; a few horses seem prone to gas colic if given inordinate amounts.

Badly made silage may be contaminated with pathogenic bacteria – *Listeria*, *Salmonella* spp. or *Clostridium botulinum* – any of which may cause problems. Some clostridial spores will inevitably be present on the ensiled crop. Horses and ponies, possibly through the absence of a rumen, are more susceptible than are ruminants to pathogenic clostridial bacteria. The toxins of *Clostridium perfringens* invoke severe enteritis and enterotoxaemia; the neurotoxins of *C. botulinum* are particularly dangerous: they cause a descending paralysis commencing with tongue and pharyngeal paresis, with dysphagia, progressive paralysis, recumbency and eventual death. These botulinum bacteria multiply in the silage clamp or bale during abnormal fermentation. It is particularly important, therefore, that clostridial growth is entirely prevented in ensiled forage intended for horses.

The fermentation of sufficient water-soluble carbohydrate in this material brings about a fall in pH, which will inhibit the growth of clostridia if the content of dry matter is at least 25%. Where the dry-matter content is only 15%, a pH of 4.5 will not inhibit the proliferation of clostridia and secondary fermentation of the silage occurs, lactic acid is degraded to acetic and butyric acids, carbon dioxide is evolved with extensive deamination and decarboxylation of amino acids, yielding amines and ammonia. The resulting rise in pH activates the less acid-tolerant proteolytic

Table 10.15 Soluble carbohydrate content of grass with a ruminant D-value of 67 (based on NIAB 1983–4a).

	Soluble carbohydrate (g/kg DM)
Italian ryegrass	31
Hybrid ryegrass	26
Perennial ryegrass (intermediate and late)	24
Perennial ryegrass (early)	23
Timothy	14
Cocksfoot	13

clostridia. The ammonia they release brings about a further increase in pH. These changes are accompanied by a characteristic smell and loss of the attractive vinegary aroma.

The likelihood of clostridial proliferation is hampered by wilting the fresh material for 36–48 hours before ensiling. In big-bale silage and haylage, dry-matter contents of 30–60% are thus achieved. Satisfactory fermentation is facilitated in normal silage, with an adequate decline in pH, by harvesting forage containing not less than 2.4–2.5% of water-soluble carbohydrate in the fresh material [the National Institute of Agricultural Botany (NIAB), Cambridge, suggest 10% in the dry matter]. In NIAB trials, where 100 kg N/ha was given in March, mean values in the first conservation cut at 67D are given in Table 10.15.

A proportion of rye grasses in any mixture obviously assists good fermentation and acid production during silage making. Less reliable material may have one or more of the following qualities:

- poor fermentation potential;
- moisture content is higher than desirable; or
- undesirable soil contamination.

If this is the case, or to avoid the growth of undesirable microorganisms and aerobic deterioration during ‘feed-out’, it may be necessary to add molasses to ensure the presence of adequate soluble carbohydrate. However, viscid liquids of this type are difficult to apply uniformly, so that additives are often used to achieve a pH of 4.2–4.6. Reliable additives are: (1) formic acid (2.3–3.0 l/1000 kg), but corrosive, and (2) cultures of *Lactobacillus plantarum* (4×10^6 cells/g fresh grass), but more expensive. Both additives properly used can lead to a more rapid fall in pH, lower content of ammonia N and acetic acid, and higher content of water-soluble carbohydrate and lactic acid than in untreated silage. Subsequent fermentation will reduce the pH further to a level that makes clostridial fermentation unlikely. If, however, a stable pH is not reached in silage with low DM content, then saccharolytic clostridia, which are present in the original crop as spores, will multiply and will initiate the secondary fermentation referred to above.

Big-bale silage, widely used in farming, and other plastic-wrapped silages have high DM content, minimal fermentation, no additives and a pH of 5–6. They are subject to aerobic deterioration, owing to lower density than bunker silage and to the potential for punctures in plastic wrappings. They depend entirely on high osmotic pressure (low water activity) to inhibit clostridial proliferation and on the absence of tears in the plastic to prevent mould growth (see Chapter 5, Plate 5.2 a–c, p. 122). Deaths have occurred among horses consuming big-bale silage associated with the presence of clostridial organisms and/or toxins, without any contemporary effects on cattle. Great care should thus be exercised in selecting silage with the appropriate aroma, a high DM content and a pH of 4.5–5.5. These are essential prerequisites of silage, or haylage, used in horse feeding if health risks are to be minimized.

Dairy cattle given extensively fermented rye grass silage lost greater amounts of N in the urine compared with those given silage of restricted fermentation, or fresh grass. Extensive fermentation, cf. restricted fermentation, provided a higher proportion of propionate and a lower proportion of acetate in the VFA of the silage. Similar comparisons have not been undertaken with horses, although it is apparent that extensively fermented herbage, or that containing significant amounts of ammonia, or n-butyric acid, is not very palatable to horses. Practice and experience are necessary adjuncts to good recipes in the achievement of the optimum product. High DM (45–60% DM), fermented, plastic-wrapped herbage is gaining in popularity and is generally more palatable to horses than is silage with a lower DM. Adult horses tend to consume more DM from a high than a low DM silage.

Metallic contamination of silage

Acidity causes the solution of heavy metals that may contaminate ensiled herbage. This is particularly important where pastures may have been used for game or clay-pigeon shooting. Extremely high concentrations of lead, causing death, have been detected by the author in silage from fields over which clay-pigeon shooting has occurred. Dissolved lead is more toxic than metallic lead and horses are seriously and permanently injured, or killed, by exposure of this kind. Other heavy metals may be similarly dissolved.

Maize silage (see also Chapter 8)

In various areas of France, and for many years, maize silage of 30–35% DM has been given to pregnant and lactating mares and to growing horses, as the main dietary constituent. Milk production is excellent and foal growth rate good. Supplemented with concentrates the silage is fairly well consumed by growing horses (Micol & Martin-Rosset 1995) and long experience has enabled breeders to use this material safely.

GRASSLAND IMPROVEMENT

Permanent pasture should be well drained and where it is low lying, or the subsoil is heavy, consideration should be given to piped drainage and, in the appropriate areas, pumped low-level ditches. Improvement in drainage reduces poaching and allows the stocking density to be increased. By lowering the water table, plant roots are encouraged to permeate lower horizons of the soil so that they gain access to a greater variety of minerals and to moisture for longer rather than shorter periods during a drought. This extends the grazing season and encourages rye grass, cocksfoot, timothy and clover, discouraging rushes, tufted hair-grass or tussock grass (*Deschampsia caespitosa*), *Agrostis*, couch, Yorkshire fog and water-tolerant meadow grasses. Fertilizers, therefore, can be more effectively used, and the increased potential growth of the resulting sward decreases the concentration of parasitic worms. It also decreases the risk of liver fluke infection. Although horses have a pronounced resistance to liver fluke, they can become infested and their economic worth is thus prejudiced. Where mixed grazing is practised, the sheep and cattle can, of course, be treated before they are introduced. The snail burden of the land may also be reduced by copper sulphate treatment or, for example, by grazing with ducks.

If piped drainage is impractical, the subsoiling of impervious heavy land will improve its structure and for a shorter period bring about the advantages of more permanent drainage. Sometimes such marginal land is infested with quite inedible species, including cottongrass (*Eriophorum* spp.), rushes and, in better-drained acid areas, by thick mats of *Nardus* grass. Here, initial improvement can frequently be effected through burning and hard grazing with steers. Most poor-quality permanent pastures can be improved by occasionally discing or heavy harrowing to break up a surface mat. This allows aeration, encouragement of better grasses, and some control of moss (*Lycopodium* spp.), mouse-ear chickweed and buttercup. In dry areas, however, little disc penetration will occur unless there has been recent rain.

Chemical control of weeds

Although the cutting of pernicious weeds such as docks (*Rumex* spp.) and thistles before they seed is helpful, it must be done on a regular basis, five or six times a season, and even then control of creeping thistle is doubtful. Thus, the use of selective weedkillers based upon methozone (MCPA) and/or 2,4-D to control broad-leaved weeds without damage to mature grass is recommended. Creeping thistles, docks and, in some upland studs, bracken encroach progressively on grazing areas. The Long Ashton Research Station, Bristol, has achieved effective control of these species with low-dose sulphonyl-urea herbicides, although these are not entirely clover-safe.

Where small prostrate weeds, such as members of the buttercup family (*Ranunculaceae*), are widespread, spraying by tractor is most convenient, but where

there are patches of creeping thistles, docks and nettles, a knapsack sprayer can be used to advantage: this causes less damage to clovers and other valuable broad-leaved herbs. Spraying should always be carried out according to the manufacturer's directions:

- (1) when there is little wind;
- (2) at a time of rapid weed growth, but before flowering;
- (3) refraining in periods of drought but avoiding rain for a few hours after spraying; and
- (4) not before newly sown pasture grasses have at least three leaves.

MCPA is effective against common nettle, daisy, dandelion, spear thistle (*Cirsium vulgare*), annual scentless mayweed (*Tripleurospermum inodorum*), black bindweed (*Polygonum convolvulus*), redshank (*P. persicaria*), cleavers (*Galium aparine*), common chickweed (*Stellaria media*), charlock (*Sinapis arvensis*), fumitory (*Fumaria officinalis*), poppy (*Papaver rhoeas*), fat hen (*Chenopodium album*) and small nettle (*Urtica urens*). It will check broadleaved dock (*Rumex obtusifolius*), bulbous buttercup (*Ranunculus bulbosus*), creeping buttercup (*R. repens*), coltsfoot (*Tussilago farfara*), creeping thistle (*Carduus arvensis*), curled dock (*Rumex crispus*), field horsetail (*Equisetum arvense*), perennial sowthistle (*Sonchus arvensis*) and soft rush (*Juncus effusus*). The most satisfactory control is achieved by spraying bulbous buttercup and chickweed in autumn and ragwort in early May. Bracken fern is very resistant to spraying, but the most effective time is August. Pastures should be left for 15 days before they are grazed in order to allow adequate time for the penetration of the herbicide, and the dead residues of poisonous weeds should be removed from the pasture. MCPA and 2,4-D are of low toxicity to animals, and pastures properly treated with them have not caused fatalities, regardless of how soon they have been grazed.

Poisonous plants

Horses and ponies do not normally graze growing poisonous weeds, but young stock or other stock freshly introduced to a pasture, following a period in stable, may eat avidly a variety of such plants. Furthermore, during times of drought, some that are deep-rooted or near watercourses and remain green entice the unwary horse. I was confronted with just such a situation in the Middle East, leading to hepatotoxicity, where the only significant amount of green herbage over a wide area was *Heliotropium europaeum* (Table 10.17). Weeds found in Europe that are poisonous to horses in both the growing and dried states are listed in Table 10.16 and those reported worldwide to be toxic to horses are listed in Table 10.17. After cutting and drying, or after being destroyed by sprays, weeds that retain their toxicity are more palatable than before and so should be removed and burnt.

Table 10.16 European plants poisonous to horses.

Species	Common name	Location	Toxic parts and effects reported
<i>Aconitum napellus</i>	Monkshood	Shady stream sides	Whole plant, especially root, very potent
<i>Agrostemma githago</i>	Corncockle	Cultivated fields	Especially seeds, saponins
<i>Anagallis</i> spp.	Pimpernel	Sandy coastal areas, cultivated land	Whole plant
<i>Aquilegia vulgaris</i>	Columbine	Damp places, lime-rich woodlands, grassland	Probably whole plant, mild hydrogen cyanide
<i>Arenaria</i> spp.	Sandwort	Dry places	Whole plant, mild
<i>Atropa bella-donna</i>	Deadly nightshade	Lowland, hedge banks, edges of woods	Whole plant, especially berries
<i>Bryonia dioica</i>	White bryony	Lowland hedges	Red berries, vegetative parts, diarrhoea
<i>Cannabis sativa</i>	Hemp	Wasteland	Flowers, fruits, low toxicity, but horses at greater risk
<i>Chaerophyllum temulum</i>	Rough chervil	Road sides	Seeds and shoots
<i>Chelidonium majus</i>	Greater celandine	Shade, banks, walls	Stems
<i>Cicuta virosa</i>	Cowbane, water hemlock	Damp places	Whole plant, especially roots, very poisonous, including hay
<i>Conium maculatum</i>	Hemlock (smooth spotted stems)	Damp places	Whole plant, mature hay unlikely to be harmful
<i>Cyclamen hederifolium</i> , <i>C. europaeum</i>	Sowbread	Woods, banks, especially southern Europe	Whole plant
<i>Daphne mezereum</i>	Mezereon	Woodlands	Berries and bark
<i>Datura stramonium</i>	Thornapple	Waste lowland	Whole plant
<i>Delphinium</i> spp.	Larkspur	Cultivated land	Whole plant, potent
<i>Digitalis purpurea</i>	Foxglove	Hedgerows, wood margins	Whole plant
<i>Equisetum</i> spp.	Horsetail	Stream banks, damp woods	Whole plant, including hay, especially horses
<i>Euonymus europaeus</i>	Common spindle tree	Hedgerows	Whole plant
<i>Frangula alnus</i>	Alder buckthorn	Woods, hedges	Leaves, bark, fruit, diarrhoea
<i>Helleborus</i> spp.	Hellebore	Lime-rich woodlands, scrub	Whole plant, potent
<i>Hyoscyamus niger</i>	Henbane	Clearings, edges of woods	Whole plant
<i>Hypericum</i> spp.	St John's wort	Grasslands	Whole plant, photosensitivity
Iridaceae	Iris, narcissus, daffodil	Gardens	Bulb
<i>Laburnum anagyroides</i>	Laburnum	Gardens, self-sown	Whole tree
<i>Lactuca virosa</i>	Acrid lettuce	Road sides	Whole plant

Table 10.16 *Continued*

Species	Common name	Location	Toxic parts and effects reported
Liliaceae (<i>Allium oleraceum</i> , <i>Endymion non-scriptus</i> , <i>Fritillaria meleagris</i> , <i>Convallaria majalis</i> , <i>Colchicum autumnale</i>)	Field garlic, bluebell, fritillary, lily of the valley, meadow saffron	Woods, meadows	Bulb
<i>Linum catharticum</i>	Purging (fairy) flax	Pastures, meadows, uplands	Seeds
<i>Linum usitatissimum</i>	Common flax, linseed	Escaped from cultivation, common	Seeds
<i>Lolium temulentum</i>	Darnel	Waste	Possibly fungal infection
<i>Lonicera xylosteum</i>	Honeysuckle	Woodlands	Berries and leaves
<i>Lupinus</i> spp.	Lupin	Light, dry soils	Whole plant, including hay, especially seeds
<i>Mercurialis annua</i>	Annual mercury	Clearings, edges of woods	Whole plant
<i>Mercurialis perennis</i>	Dog's mercury	Poor land	Whole plant
<i>Oenanthe crocata</i>	Hemlock, waterdropwort	Damp places	Whole plant, especially roots
<i>Papaver</i> spp.	Poppy	Cultivated land, wasteland	Whole plant
<i>Paris quadrifolia</i> (Liliaceae)	Herb Paris	Damp alkaline woodland	Low toxicity
<i>Pteridium aquilinum</i>	Bracken fern	Heaths, wasteland, woods	Whole plant, including rhizome and hay
<i>Ranunculus</i> spp., <i>R. sceleratus</i>	Buttercup, celery-leaved buttercup	Damp places, pastures	Whole plant, irritant, colic, inflammation
<i>Rhamnus catharticus</i>	Common (purging) buckthorn	Chalky thickets	Fruit, diarrhoea
<i>Rhododendron</i> spp., <i>Azalea</i> , <i>Kalmia</i>	Rhododendron, azalea, kalmia	Woodland	Whole plant
<i>Saponaria officinalis</i>	Soapwort	Hedges, banks, damp places	Whole plant, mild, saponins as for <i>Stellaria</i>
<i>Senecio</i> spp.	Especially ragwort	Old pasture	Whole plant
<i>Solanum dulcamara</i>	Woody nightshade, bittersweet	Road sides	Whole plant
<i>Solanum nigrum</i>	Black nightshade	Cultivated lowland	All parts, especially berries
<i>Solanum tuberosum</i>	Potato	Cultivated land	All parts including tubers when damaged
<i>Stellaria</i> spp.	Chickweeds	Ubiquitous annual	Whole plant, mild
<i>Tamus communis</i>	Black bryony	Woods, hedgerows	Possibly berries, mild toxicity
<i>Taxus baccata</i>	Yew	Woods, gardens	Whole tree, very potent

Table 10.17 Plants reported in the scientific literature to have caused poisoning in horses and ponies (Hails & Crane 1982).

Species	Common name	Effects reported
<i>Agrostemma githago</i>	Corncockle	Hypersalivation, abnormal thirst, accelerated respiration
<i>Allium validum</i>	Wild onion	Haemolytic anaemia
<i>Amsinckia intermedia</i>	Tarweed	Walking disease, haemolytic anaemia, liver necrosis, possibly nitrate toxicity
<i>Aristolochia clematitis</i>	Birthwort	Tachycardia, weak pulse, decreased appetite, constipation, polyuria
<i>Arum maculatum</i>	Lords-and-ladies, Cuckoopint	Purgative, irritation of GI mucosa and kidneys, fatty liver
<i>Astragalus</i> spp.	Locoweed or milk vetch	Eye lesions, locoism, Se accumulation in several species
<i>A. lentiginosus</i> , <i>A. mollissimus</i>	Locoweed	Depression, anorexia, ataxia, hyperexcitability and especially violent responses to stimuli; effects irreversible
<i>Atalaya hemiglauca</i>	Whitewood	Anorexia, dullness, irritability
<i>Centaurea repens</i>	Russian knapweed	Nigropallidal encephalomalacia, severe damage to nerve cells, muscular hypertonia
<i>Centaurea solstitialis</i>	Yellow star thistle	Nigropallidal encephalomalacia (chewing disease), hypertonia of muscles and muzzle
<i>Cestrum diurnum</i>		Calcinosis hypercalcaemia (other <i>Cestrum</i> spp. cause severe gastritis and liver degeneration, but not reported in horse)
<i>Coriandrum sativum</i>	Coriander	(No signs given in Hails & Crane 1982)
<i>Crotalaria</i> spp.		Hepatotoxicity, jaundice, dyspnoea, weak pulse, collapse
<i>C. crispata</i>		Kimberley horse disease, anorexia, dullness, staggering gait
<i>C. dissitiflora</i> var. <i>rugosa</i>		(No signs given in Hails & Crane 1982)
<i>C. retusa</i>		Kimberley horse disease, liver and central nervous system lesions, anorexia, dullness, staggering gait
<i>Cuscuta</i> spp.	Dodder	Enteritis, anorexia, nervous symptoms
<i>C. campestris</i>	Peppery cuscus, dodder	Not described, but staggering, salivation, increased pulse and respiratory rates reported in cattle
<i>C. breviflora</i> ,		
<i>Echium lycopsis</i> (or) <i>E. plantagineum</i>)	Purple viper's bugloss	Hepatotoxicity, blindness
<i>Equisetum</i> spp.	Horsetail, maretail	Thiamin deficiency – the horse is highly susceptible
<i>E. fluviatile</i>	Water horsetail	
<i>E. hyemale</i>	Rough horsetail or Dutch rush	Akin to bracken poisoning owing to presence of thiaminase
<i>E. palustre</i>	Marsh horsetail	Loss of condition, some diarrhoea, usually caused by eating hay containing horsetails – administer thiamin or dried yeast
<i>E. variegatum</i>	Variegated horsetail	

Table 10.17 *Continued*

Species	Common name	Effects reported
<i>Eupatorium</i> spp. (incl. <i>E. rugosum</i>)	Including white snakeroot and hemp agrimony	Trembling, lethargy
<i>Eupatorium adenophorum</i>	Croftonweed	Pulmonary oedema, possible nitrate toxicity
<i>Galeopsis</i> spp.	Hempnettle	Pulmonary oedema, enteritis, anorexia
<i>Glyceria maxima</i>	Reed sweet-grass	Cyanide poisoning
<i>Helichrysum cylindricum</i>	Everlasting flower	Blindness, encephalopathy
<i>Heliotropium europaeum</i>	European heliotrope	Hepatotoxicity
<i>Hypericum crispum</i>		Photosensitivity
<i>Indigofera dominii</i> , <i>I. enneaphylla</i>	Birdsville indigo	Birdsville disease, drowsiness, immobility, discharges from eyes and nose, dragging of hind feet, liver lesions
<i>Juglans nigra</i>	Black walnut	Acute laminitis
<i>Jussiaea (Ludwigia) peruviana</i>		
<i>Lathyrus nissolia</i>	Grass vetchling	Incoordination and collapse during exercise
<i>Lupinus</i> spp.	Lupin	(1) Chronic: lupinosis-congenital defects, liver damage (due to mould growing on lupins); (2) acute: respiratory paralysis due to lupin alkaloids when large amounts of the plant eaten
<i>Medicago sativa</i>	Lucerne	Photosensitization, possible oestrogen toxicity; certain strains cause anaemia and hepatotoxicity
<i>Morinda reticulata</i>		Se toxicity
<i>Nerium oleander</i>	Oleander	Highly toxic – convulsions, diarrhoea, colic, petechial haemorrhages
<i>Oxytropis campestris</i> , <i>O. sericea</i>	Locoweed	Nervous signs, anorexia, ataxia; damage permanent
<i>Papaver nudicaule</i>	Iceland poppy	Ataxia, convulsions
<i>Perilla frutescens</i>		Lung disease
<i>Persea americana</i>	Avocado	(No signs given in Hails & Crane 1982)
<i>Phaseolus vulgaris</i>	Kidney, haricot or navy bean	Central nervous system lesions, GI disorder
<i>Pimelea decora</i>	Rice flower	Colic, diarrhoea, collapse, ulceration of mouth, tongue, oesophagus, gastritis (also St George disease in cattle)
<i>Polygonum aviculare</i>	Knotgrass, wireweed	Nitrite poisoning, GI irritation
<i>Prunus laurocerasus</i>	Cherry laurel	Cyanide poisoning (also caused by other <i>Prunus</i> spp. e.g. <i>P. serotina</i> – wild black cherry)
<i>Pteridium aquilinum</i>	Bracken	Thiamin deficiency
<i>Quercus</i> spp. (incl. <i>Q. rubra</i> var. <i>borealis</i>)	Oak	Acorn poisoning: hepatotoxicity; oak-leaf poisoning: dullness, diarrhoea, constipation, dark urine
<i>Ricinus communis</i>	Castor-oil plant	Castor-bean poisoning: dullness, incoordination, sweating, tetanic spasms, watery diarrhoea
<i>Robinia pseudo-acacia</i>	False acacia	Acacia bark poisoning: colic, diarrhoea, irregular pulse, hyperexcitability, paralysis

Table 10.17 *Continued*

Species	Common name	Effects reported
<i>Senecio</i> spp.	Groundsel, ragwort	Hepatotoxicity
<i>S. jacobaea</i>	Common ragwort	Hepatotoxicity
<i>Setaria sphacelata</i>	Bristle grass	Oxalate poisoning, osteodystrophia fibrosa
<i>Sinapis arvensis</i>	Charlock	Cyanide poisoning, colic, gastroenteritis, diarrhoea
<i>Solanum malacoxylon</i>		Calcinosis – wasting, stiffness, hypercalcaemia, calcification of arteries
<i>Sorghum almum</i>	Sorghum	Cystitis, ataxia (growing grass)
<i>Sorghum bicolor</i>	Sudan grass	Cystitis, ataxia (growing grass)
<i>Sorghum halepense</i>	Johnson grass	Cyanide poisoning
<i>Sorghum sudanense</i>	Sudan grass	Possible lathyrism, equine cystitis and ataxia, ankylosis (growing grass)
<i>Sphenosciadum capitellatum</i>	Whitehead	(No signs given in Hails & Crane 1982)
<i>Stipa viridula</i>	Sleepy grass	Hypnosis
<i>Swainsona</i> spp.	Darling pea	Depression, emaciation, ataxia, blindness
<i>Tanaecium exitiosium</i>		Weakness, staggering, collapse, frequent micturition, inflammation of stomach and heart
<i>Taxus baccata</i>	Yew	Heart failure – trembling, dyspnoea, collapse
<i>Taxus cuspidata</i>	Japanese yew	Heart failure – trembling, dyspnoea, collapse
<i>Trichodesma incanum</i>		Severe liver damage in horses and other farm stock
<i>Viscum album</i>	Mistletoe	Incoordination, dilated pupils, salivation, hypersensitivity

Reseeding

If there is little pressure on available grazing land and the appropriate equipment is available, it can make economic sense to reseed worn-out pastures after ploughing, as long as the land is potentially fertile. No permanent improvement would, however, result unless drainage, ditching, liming, fencing and hedging are first put in order. If there are large areas of pernicious perennial weeds in the old turf, they should first be destroyed with herbicides and two to three weeks should elapse before ploughing begins. For spring sowing, autumn ploughing of the old turf is desirable. Sometimes it may be necessary to use heavy discs or cultivators to break up the turf. Also, there is some justification for killing all the old turf by spraying with glyphosate, delaying ploughing for at least two weeks to ensure root kill. On very light soils it may be possible to plough during the late winter and sow in March or April in the northern hemisphere, but it is essential to achieve consolidation of the ground and a fine tilth after ploughing.

A pasture-seed mixture should be sown as soon as a good seedbed can be effected, before the surface soil is subjected to periods of desiccation. In the UK,

Table 10.18 Suggested seed mixtures and minimum seeding rates (kg/ha) for permanent pastures when a good seedbed has been established (under adverse conditions the quantities may need to be increased up to double these amounts).

	kg/ha
<i>Seed mixture 1</i>	
Perennial rye grass (Melle ¹ or Contender)	10.00
Perennial rye grass (Talbot, Parcour or Morenne)	3.50
Smooth meadow-grass ² (Arina, Dasas)	0.75
Creeping red fescue (Echo)	2.00
Rough meadow-grass (VNS)	0.75
White clover (Blanco or NZ Huia)	0.75
Total	17.75
<i>Seed mixture 2</i>	
Perennial rye grass (Trani or Springfield)	10.00
Perennial rye grass (Melle)	5.00
Timothy (S48, S352, S4F)	2.00
White clover (Milkanova)	2.00
Total	19.00

¹ A mixture of strains similar to S23 and S24 could be used to give a range of heading dates.

² Kentucky bluegrass or smooth meadow-grass (*Poa pratensis*).

sowing should be accomplished by March or April, otherwise a delay until July or August may be inevitable, and undertaken then only if there are prospects of rain. It is also imperative that plant foods be provided: Table 10.10 gives suggested rates of application. In areas of low rainfall, seeds should be sown 2.5 cm (1 in) deep; in areas of higher rainfall the sowing depth should be about 1.2 cm (0.5 in). Drilling deeper than 2.5 cm, which would be advisable in the height of summer, will result in seedlings of small seeds, such as poas and clovers, not reaching the surface, therefore for reasons of economy, these should be omitted from the seed mixture. A fine firm tilth is then produced by harrowing and rolling in order to avoid a puffy, rapidly drying surface soil. Two seed mixtures are suggested in Table 10.18. These are composed of persistent strains that should not be given a cover crop and which suffer more than quicker-growing strains from drought in the seedling stage if they have been sown in late spring in poorly formed seedbeds.

In formulating one's own seed mixtures, the smaller the seed the lower the weight required per hectare. Thus, a lower weight of rough meadow grass, timothy and wild white clover would be required than of perennial rye grass or meadow fescue and less is required of persistent leafy tillering strains of grasses for permanent pastures than would be used of aggressive strains in short-term leys. The better late-flowering strains of rye grass and timothy should not be mixed with aggressive early-flowering strains in the establishment of a permanent pasture. On light soils, smooth meadow

grass or Kentucky bluegrass (*Poa pratensis*) may be a helpful constituent of the mixture by virtue of its underground stems, which knit the surface soil together. On heavier soils this species might be replaced with tall fescue.

For some time a new sward will be more susceptible to destruction by treading than would an established turf and so ideally it should not be grazed in the first year, but should nevertheless be topped to prevent the grasses and any annual weeds from flowering. If the latter are present in excessive quantities, a selective weedkiller could be used, although this would check the establishment of clover, and in any event should not be used before the grasses have developed two or three leaves. Another option might be grazing by sheep late in the first year, but not when the surface is wet. A hay crop should not be taken from a newly established permanent pasture during the first two years at the very least.

Where ploughing and reseeding are impractical, or it is essential to establish a new pasture quickly, the author has had considerable success in horse paddocks by spraying the old turf with glyphosate once or twice during a period of rapid growth and leaving it for at least 14 days. Following this, there are two alternative procedures for reseeding:

- (1) The surface is thoroughly disced (Plate 10.2), harrowed and rolled to effect a good tilth and seedbed before drilling the seed. This is delayed until the surface trash has decayed and if there are large amounts it can be burnt or raked off. The essential point is that seedlings should be surrounded by mineral soil in a firm seedbed. Burning will destroy seeds of exotic plants that may have remained dormant in permanent pastures for many years and that can yield a fascinating, but mildly bothersome, crop in the young sward. Moreover, excessive vegetative matter, which may contain some herbicide residues, can suppress the growth of the seedlings, but the cultivations allow soil microorganisms to contact and rapidly destroy residual herbicide. In areas of low rainfall the cultivations will dry out the surface soil during the summer, which also suppresses seed germination.
- (2) After the vegetation has died, therefore, surface moisture is retained and time is saved by direct drilling the seed immediately into the soil with a special drill that has heavy disc coulters for cutting through the dead turf. The same principles of top dressing and rolling at the appropriate times should be applied as for the more traditional husbandry described.

Another alternative is to graze a worn-out paddock intensively with steers, then disc and harrow heavily at a time when the surface soil is sufficiently moist to allow penetration of the implements. After a reasonable seedbed has been attained, sow, harrow, roll and fertilize as before. Heavy regrowth of the old turf can be controlled by regular topping until the new seedlings are well established. This procedure is the least expensive but unlikely to give complete satisfaction, especially where the previous sward was dense or its regrowth too rapid during the seedling stage.

(a)



(b)



(c)



Plate 10.2 Preparation of soil for resowing permanent pasture at Upend Stud, Newmarket, Suffolk. Discing began eight days after spraying with glyphosate, a satisfactory seedbed was achieved 21 days after spraying and seed was drilled after 24 days, followed by rolling. Pictures (a) and (b) show stages in seedbed preparation and picture (c) shows pasture one year after sowing. Previously this paddock was rife with dock and thistles.

Plant disease control

Grass seedlings are sometimes killed soon after germination by soil-borne *Fusarium culmorum*. There is increasing reluctance to use pesticides that may be harmful to useful domestic insect and other animal species. Success has been achieved in the control of this fungus by using another fungus that is antagonistic to the *Fusarium* but harmless to the grass seedlings. Indeed, a number of endophyte species (living entirely within the plant tissue and producing no spores) have been discovered that are synergic with grasses, that is, they even promote the health and growth of the grass species. Endophytes occur in both rye-grass and fescue pastures (see also 'Equine dysautonomia (EGS, grass sickness)', and 'Fescue toxicosis' caused by *Acremonium coenophialum*, this chapter). Endophytes are transmitted in infected seed and the resulting plants may be more resistant to a range of pests, grow more vigorously, producing a higher DM yield, and may even be more drought tolerant and winter hardy. Nevertheless, considerable care is now exercised by plant breeders in the use of these fungi as there is confirmed evidence that certain endophytes are a cause of disease in grazing stock. Rye grass is frequently infected by *Acremonium lolii* which is likely to be the cause of sporadic disease in grazing animals (see 'Rye-grass staggers', this chapter), associated with unthriftiness and loss of coordination, observed in the UK, New Zealand and in several other countries, and *A. coenophialum* is the cause of fescue toxicity. Interestingly, it is thought that the tremorigenic toxin causing the animal disease is different from that afflicting insect pests, so useful progress should be possible in selecting endophyte strains for inoculating seeds that are benign to four-footed stock. Only time will tell.

TROPICAL GRASSLAND AND FORAGES

A characteristic of most tropical forages is a high yield of dry matter, which is relatively rich in fibre but impoverished of crude protein and P (Table 10.19) and from which the rewards of animal production are relatively meagre. There are, nevertheless, large differences between the products of wet and dry seasons. In one study (Kozak & Bickel 1981) the crude protein content of grasses decreased from a range of 6–10% in the rainy season to 4–5% in the dry season in Tanzania without any change in the crude fibre content, yet apparent digestibilities of the crude protein were 34–58% and 16–25%, respectively. Kozak & Bickel also noted that the digestibility of the dry matter of pasture forage decreased from 47–63% at heading to 30–53% at flowering.

It has been asserted that the yield per hectare of digestible nutrients is at best only half what can be achieved from temperate grassland. The low digestibility of tropical grass is apparently an effect of a higher lignin content than that of temperate grass. Analyses reveal that as the environmental temperature rises there is a fall in the cellulose content of tropical grass but a proportionate rise in the hemicellulose and lignin contents. Malaysian experience has shown that horses fed as much cut tropical grass as they can eat, plus oats or other concentrates, lose weight. Their general

Table 10.19 Dry-matter composition of four tropical grasses fed to horses compared with a temperate (Newmarket, UK) grass sample (D. Frape unpublished observations).

	Young Napier grass	Napier hay	Mature Napier hay	Signal grass	Signal grass hay	Paragrass hay	Guatemala grass	Newmarket grass
Crude protein (%)	5.7–10	5–6	2.7–2.9	11–11.5	5–6	5.4–9.8	7.4–7.5	12.7
MAD fibre (%)	43–51	54–55	54–55	41–42	51–52	42–44	46–47	27.5
Ash (%)	3.2–4.5	3.6–3.8	2.1–2.3	7.3–7.8	3–3.2	3.4–8	6–7	9.95
Ca (%)	0.046–0.16	0.08–0.1	0.1–0.12	0.09–0.13	0.09–0.1	0.11–0.2	0.05–0.06	0.93
P (%)	0.06–0.16	0.11–0.13	0.1–0.12	0.17–0.18	0.14–0.15	0.15–0.23	0.17–0.175	0.31
Mg (%)	0.21–0.32	0.20–0.22	0.22–0.25	0.26–0.27	0.11–0.12	0.18–0.36	0.087–0.088	0.16
K (%)	0.9–1.3	0.8–0.9	0.38–0.4	2.8–3.3	0.95–1.05	0.66–2.6	2.1–2.2	1.9
Na (mg/kg)	170–690	200–300	170–190	510–650	170–180	1280–11300	150–170	1800
Zn (mg/kg)	21–38	34–36	20–22	27–29	—	35–43	—	38
Se (mg/kg)	0.055	—	—	—	0.03	0.03	0.04	0.1
F (mg/kg)	8.4	—	—	—	—	—	—	—

MAD, modified acid-detergent.

health and their performance can be improved by limiting their access to grassland, or its products, and confining them for greater periods of time to the stable, which affords shade, cool ventilation and a more satisfactory control of insect-borne disease. Furthermore, where the land is light, significant amounts of sand may be consumed by grazing stock and this may interfere with bowel function.

Plate 10.3a shows TB paddocks at 1350m in Zimbabwe at the end of October, during the foaling season (surprisingly the hottest part of the summer, before the rains come). The grass is dry and bleached and the only green material appears to be *Indigofera* spp., Leguminosae, probably *I. setiflora* (Plate 10.3b). Species of this genus contain a hepatotoxic amino acid, indospicine, which competes with arginine, causing liver and kidney damage (Birdsville disease). Horses may be protected from Birdsville disease in Australia by supplementing their feed with arginine-rich substances such as peanut meal or gelatine. Signs of toxicity include loss of appetite, apathy, discharges from the eyes and nose, loss of flesh, laboured breathing and severe incoordination, with a dragging of the hind feet. The horse may fall backwards during a canter. Many other potentially harmful plant species were identified in and around these TB paddocks (Table 10.20).

TB studs in Zimbabwe give mares 9kg (barren mares) to 12kg (lactating mares) relatively high protein concentrates daily. This will also reduce the waste heat of fermentation which would be generated should otherwise large amounts of roughage be given. Another reason for the large concentrate allowances may be not only the low digestibility of the herbage, but also a recognition of adverse reactions if large amounts of herbage containing hazardous plants are consumed.

Oxalate poisoning

(See also 'Grass toxins', this chapter.) In the author's experience, working horses introduced to the tropics and required to subsist on indigenous forages and cereal grains frequently exhibit decreased performance, lameness (changing from one leg to another and involving creaking hip and shoulder joints), arching of the back, swelling of the facial bones and muscular wasting of croup and rump. Similar observations and the poisoning of grazing cattle have been recorded in various parts of south-east Asia, the Philippines, Brunei and north Australia (Seawright *et al.* 1970; Blaney *et al.* 1981a,b).

The main forage crops grown for horses in Thailand are called locally 'Yakon' and 'Mauritius Grass'. Some Purple Guinea, Rhodes and Star Grass are also grown, but almost no legumes. These grasses contain excessive oxalates and low levels of available Ca and apparently of Cu. Frequent cases of OCD and wobbler syndrome are presented, and a trace-element supplementation is often helpful (Wood 2001, personal communication). The poor nutritional quality of tropical grass has frequently been overcome by the introduction of high-quality clovers, trefoils and *Medicago* spp., including lucerne, selecting those varieties low in tannins, as increased tannin concentrations are prevalent when legumes are grown on tropical soils deficient in P.



(a)



(b)

Plate 10.3 TB stud in Zimbabwe, 1350m, during October. This is the foaling season and the hottest and driest month, when pastures can be of poor feeding value: (a) a general view of the paddock; (b) the only green herbage in the paddock, *Indigofera* spp., a legume said to contain substances toxic to ruminants.

Table 10.20 Hazardous plant species identified in and around paddocks in a subtropical TB stud.

Plant species	Comments
<i>Eleusine indica</i> , <i>Panicum novemnerve</i>	Thyrotropic cyanogenic glycosides
<i>Setaria pumila</i> , <i>Portulaca</i> spp., <i>Oxalis</i> spp.	Oxalates reducing bone calcification
<i>Verbena bonariensis</i>	Members of the <i>Verbena</i> family contain hepatotoxic icterogenin and other triterpenoids, e.g. rehmannic acid, identical to that causing <i>Lantana</i> poisoning: 'Beach disease' (see below)
<i>Nicandra physalodes</i>	A poisonous member of Solanaceae
<i>Senecio venosus</i>	Members of the genus <i>Senecio</i> contain hepatotoxic alkaloids
<i>Sideranthus</i> spp.	Se accumulators*
<i>Amaranthus hybridus</i>	Nitrates and the alkaloid lycorine, salivation, diarrhoea, paralysis
<i>Richardia scabra</i>	(False ipecacuanha) Very poisonous
<i>Tagetes minuta</i>	Very poisonous, roots poisonous to nematodes
<i>Spermacoce senensis</i>	Very poisonous
<i>Lantana camara</i>	Very poisonous, rehmannic acid, a hepatotoxin, 'Beach disease': cholestasis, anorexia, haemorrhagic gastroenteritis, ataxia, photosensitization
<i>Convolvulus sagittatus</i> var. <i>aschevsonii</i>	Very poisonous, purgative, nervous signs, incoordination, tremors, pulmonary oedema, polyuria, blindness; contains lysergic acid alkaloids
<i>Cynodon dactylon</i>	(Bermuda grass) Hepatotoxins causing secondary photosensitization
<i>Pennisetum clandestinum</i>	(Kikuyu grass) Salivation, thirst, distension and inflammation of GI tract, incoordination etc.
<i>Polygala albidula</i>	Members of the genus <i>Polygala</i> cause tremors, gastroenteritis, incoordination, cerebral congestion
<i>Leucas martinicensis</i>	Tetracyclic polyol andromedotoxin causes hypotension, respiratory depression, attempts to vomit. The amino acid mimosine, a depilatory, causes haemorrhagic gastritis in horses
<i>Indigofera</i> spp.	Hepatotoxic amino acid, indospicine, causing liver and kidney damage: 'Birdsville disease'

*Over 2mg Se/kg was detected in representative pasture herbage samples.

The signs described above are those of osteodystrophia fibrosa (big-head), found by the author (unpublished observations) and others (Blaney *et al.* 1981a) to be caused by large amounts of oxalate and small amounts of Ca and P in many tropical grasses. Most published reports have concerned species of the genus *Setaria* (Blaney *et al.* 1981b; Seawright *et al.* 1970), which may contain 30–70g oxalate/kg DM. A simple method for the quantitative estimation of oxalate in tropical grasses has been proposed by Roughan & Slack (1973). Lesser but harmful quantities of oxalates have been found in the widely used Napier (*Pennisetum purpureum*) and Signal (*Brachiaria* spp.) grasses and Paragrasses. For several reasons Napier is an inferior grass for horses. Some chemical characteristics of each of these tropical grasses, for comparison with temperate grasses, are given in Table 10.19. The samples were shown to be deficient also in Se. Ponies given a diet containing 10g oxalic acid/kg

plus 4.5 g Ca/kg exhibit decreased urinary Mg and are in negative Ca balance through reduced Ca absorption (Swartzman *et al.* 1978). It has been concluded that problems in horses may arise where the dietary dry matter contains more than 5 g total oxalates/kg with a Ca:oxalate ratio of less than 0.5 (Blaney *et al.* 1981a). Horses presenting typical signs, and which were in negative balances of both Ca and P, supplemented once per week with limestone, rock phosphate or dicalcium phosphate and 50–60% molasses, consumed up to 200 g Ca and 50 g P per hour and changed to positive Ca and P balances (Gartner *et al.* 1981). Success has also been achieved by supplementing horses with 125 g limestone per day (providing 45 g Ca) until the initial abnormality has subsided and then reducing the amount to 100 g daily. If rock phosphate is introduced to the diet, the F intake should not be allowed to exceed 50 mg/kg of the total diet.

POISONOUS PLANTS

Poisonous plants are generally unpalatable. They may be consumed when there is feed scarcity, when the horse has not previously encountered them, or when present dried in hay. Poisonous-weed seeds/moulds, as contaminants of grains, may include *Lolium temulentum*, *Ricinus communis* and *Claviceps purpurea*. Table 10.16 lists the plant species more likely to be a cause of horse illness in Europe.

Plant toxicosis caused by associated microorganisms in grasses

There are several microbial species growing in plants that are a cause of intoxication in horses. The subject of microbial toxins has received much attention in recent years, probably as a result of the development of laboratory methods for their analysis.

Fescue toxicosis

Perhaps one of the most pressing syndromes, at least in the USA, has been equine fescue toxicosis. Approximately 688 000 horses in the USA are kept on tall fescue (*Festuca arundinacea*) pastures, and for many years there have been reports of reproductive problems in mares grazing this species (Cross *et al.* 1995). Signs include increased gestation length, agalactia, foal and mare mortality, tough and thickened placentas, dystocia (abnormal labour at foaling), weak and dysmature foals, increased sweating during warm weather, reduced serum prolactin and progesterone and increased serum oestradiol-17 β concentrations. [Apparently, endophyte-infected tall fescue pastures suppress serum progesterone in late gestation, but fail to suppress serum progesterone, or T₃ or T₄ concentrations in early- or mid-gestation (Hill *et al.* 2001).] Unlike the effects in many other species, horses consuming infected tall fescue do not exhibit increased body temperature.

Table 10.21 Ergot profiles in endophytic fungi isolated from grasses growing in central Georgia, USA (Bacon 1995).

Ergot alkaloids	<i>Balansia</i> spp.		<i>Acremonium coenophialum</i>
	<i>B. epichloe</i>	<i>B. henningsiana</i>	
Chanoclavine I	+	+	–
Isochanoclavine I	+	+	–
Agroclavine	+	–	+
Elymoclavine	+	–	+
Penniclavine	+	–	+
Ergoclavine	+	–	–
Ergonovine	+	–	±
Ergonovinine	+	–	–
6,7-Secoagroclavine	+	–	+
Dihydroelymoclavine	–	+	–
Festoclavine	–	–	+
Ergovaline	–	–	+
Ergovalinine			+

The abnormalities in gravid mares are caused by vasoconstrictive ergot peptide alkaloids (pyrrolizidine alkaloids have also been isolated) produced by endophytic fungi, principally *Acremonium coenophialum*, but also by *Balansia epichloe* and *B. henningsiana* identified in tall fescue (Table 10.21). These fungi infect other warm season weeds and grasses (including *Agrostis*, *Andropogon*, *Eragrostis*, *Paspalum*, *Sporobolu* and *Stipa*), in none of which, however, are they pathological to the plant. Recent evidence showed that ergovaline, the primary ergopeptide isolated from *A. coenophialum*, at a dietary concentration of up to 308 µg/kg caused no adverse effect on nutritional or reproductive performance of mares.

Endophyte-infected tall fescue hay is less digestible than endophyte-free hay, and young horses consuming infected pasture grow more slowly than do those on endophyte-free pasture. The alkaloids are apparently serving as D₂ dopamine receptor agonists, explaining their prolactin-lowering effect. Cross *et al.* (1995) advise that domperidone, a dopamine receptor antagonist, is effective in preventing the signs of tall fescue toxicosis in horses without neuroleptic side effects. The minimum effective dose in gravid mares is 1.1 mg/kg BW daily given orally for 30 days before foaling, or 0.44 mg/kg BW given subcutaneously for ten days before foaling.

Fescue toxicosis causes increased susceptibility to high environmental temperatures and light intolerance (Porter & Thompson 1992), and so recent hot summers in Europe may have increased the frequency there of this disease in horses, cattle and sheep. *Acremonium* grass endophytes are taxonomically aligned with the family Clavicipitaceae and they live their entire life within the aerial parts of their grass host, producing no spores.

Rye-grass staggers

Acremonium lolii is the endophyte of perennial rye grass (*Lolium perenne*), which produces the indole-isoprenoid alkaloids paxilline and lolitrem (Porter 1995), causing rye-grass staggers, a neurological condition characterized by incoordination, staggering, head shaking and collapse in horses that have been disturbed. Lolitrem is in highest concentration in the leaf sheaths and lowest in the leaf blades. Thus, staggers is most frequently observed in closely grazed pastures and it has been reported in horses eating rye-grass straw. Reports from New Zealand and Oregon, USA, are the most frequent. The ergopeptine alkaloids produced by this endophyte may be a cause of poor growth and poor reproduction on rye-grass pastures. The subject of the effect of natural toxins on reproduction in livestock has been reviewed by James *et al.* (1992) and by Cheeke (1995).

Pasture treatment against Acremonium spp.

At present there seems to be no effective prophylactic treatment for the pastures in which tall fescue or rye grass is infected with *Acremonium* spp. The pasture infection improves the vigour and persistency of these grass species. In New Zealand, prevention is afforded by protecting the grass against weevil damage, and plant breeding is likely to be the route by which the problem is overcome. At present, by virtue of the increased vigour endowed, endophyte-infected cultivars of both rye grass and tall fescue make up an increasing share of the total seed production.

Sleepygrass toxicosis

Sleepygrass (*Stipa robusta*) is a perennial bunch grass found on rangelands of the southwestern USA. Consumption of the grass causes a profound stuporous condition which may last for several days in horses. *Acremonium* endophytes, containing the ergot alkaloid lysergic acid amide, are probably the causative agents (see 'Fescue toxicosis', above).

Zearalenone

It has been proposed that a foetal-loss syndrome facing Kentucky's pregnant mares and causing poor foal growth was caused by the *Fusarium* toxin zearalenone in the pasture and hay at concentrations of 0.1–0.3 mg/kg. A feed-additive mycotoxin-binding agent that prevents absorption of zearalenone in the GI tract is now available commercially in Kentucky.

Vomitoxin

Forage samples in southwestern Ontario were examined for the *Fusarium* toxins vomitoxin, zearalenone and T2 toxin. Vomitoxin, at concentrations exceeding

4mg/kg DM, is associated with loss of appetite, vomiting, lesions of the intestinal tract, immunosuppression, lethargy, and ataxia. Raymond *et al.* (2001) found that 22% of forage samples contained 2–4mg/kg DM. Wright *et al.* (2003) detected low concentrations of ergot alkaloid and fusarium mycotoxin contamination of hay and straw to which mares were exposed in Ontario, but found no relationship with several reproductive parameters. The level of contamination in most 'presentable' hay samples appears to be without ill-effect.

Annual rye-grass toxicosis

Annual rye-grass toxicosis occurs particularly in South Africa and Australia and is caused by a group of highly toxic glycolipids called corynetoxins. The signs are neurological disturbances, presented as high stepping gait, ataxia and convulsions. It is a lethal condition causing damage to the cerebellum. The aetiology is complex. Seedlings may become infected by a soil nematode, *Anguina agrostis*, that leads to a gall in the flower, where the worm lays eggs. The nematode is nontoxic, but if it is, in turn, infected by a bacterium, *Clavibacter toxicus*, corynetoxins are produced and the galls are then toxic. There is some evidence that the toxin is produced only if the bacteria are themselves infected with a bacteriophage. Control of the toxicosis requires that nematode infection of the grass is prevented. Crop rotation, field burning, clipping immature seed heads, fallowing and avoidance of transfer of infected material to other fields are control methods.

Equine dysautonomia (EGS, grass sickness)

Grass sickness is now recognised in England, Scandinavia, Switzerland, the Falkland Islands and Patagonia, but rarely in North America, Australia or Ireland. Clinical and post-mortem findings in the coeliacomesenteric ganglia of horses grazing Chilean pastures at near freezing temperatures, indicated they had been suffering from grass sickness, described locally as *mal seco* (Araya *et al.* 2002). *Clostridium botulinum* was first implicated as a causative agent in EGS when it was isolated from the GI tract of a horse with the disease in 1919. As early as 1923, Tocher suggested that EGS was a toxicoinfection and he applied a *C. botulinum* toxin/antitoxin mixture to vaccinate horses. This was shown to give protection and to reduce mortality. Clostridial bacteria can multiply in soil under anaerobic conditions and so a clostridial cause is consistent with grazing by adult horses. This is especially true when the grazing venue is changed, possibly causing an upset to the gastrointestinal milieu and its protective microflora. It is speculated that the reason other horses in contact with EGS cases seem to be at lower risk is that they ingest a non-infectious dose of the organism and develop immunity. Recently, horses with EGS have been found to have lower levels of systemic IgG to both surface antigens of the type C neurotoxin, but horses previously in contact with EGS, or which had grazed land where it had occurred, revealed higher levels of antibodies to these antigens (Hunter & Poxton 2001). This may indicate the potential for vaccination.

Classical botulism is associated with dysfunction of the neuromuscular junction and death is usually associated with respiratory failure. There are no consistent histopathological lesions in any tissue. In contrast, EGS is characterised by profound neuronal destruction in the autonomic, and particularly the enteric, nervous system. Clinical signs include muscular tremors, patchy sweating, difficulty in swallowing, salivation, dilation of the bowels and stomach and chronic loss of weight. It is recognised that *C. botulinum* causes two distinct clinical syndromes in man: that resulting from the ingestion of the toxin, and infant botulism, in which the organism replicates in the gut, releasing toxin that is absorbed through the gut wall. This is known as a toxicoinfection and it has been proposed as a cause of EGS. There are eight antigenically distinct types of toxin, but most equine botulism is caused by *C. botulinum* types B and C. Type C, found in EGS, uniquely produces three neurotoxins that may cause neurotoxicity and neurodegeneration as well as interference with neuromuscular transmission. In the last five years, Hunter *et al.* (1999) adduced evidence that *C. botulinum* type C and the neurotoxin (BoNT/C) were present in the ileum of nearly half the cases of EGS they investigated, but in only 7% of controls.

Further environmental agents may augment the risk. Highly cyanogenic clover is abundant in EGS fields and B.C. McGorum (personal communication) proposed that the ingestion of this clover, containing the cyanogenic glycosides linamarin and lotaustralin, may contribute to the neurotoxicity of EGS. Moreover, the detoxification of these substances depletes plasma sulphur amino acids, found to be below normal in EGS cases (McGorum & Kirk 2001). Before EGS horses had become anorexic, McGorum concluded, they would probably reveal high concentrations of blood cyanide and of plasma and urinary thiocyanate. Recent evidence has revealed elevated plasma dihydroxyphenylalanine (DOPA) levels in EGS horses (McGorum *et al.* 2003), probably reflecting a disturbance in catecholamine metabolism. Clearly much is yet to be revealed about EGS and its control, and conclusions should not be drawn prematurely concerning the cause(s).

Chronic lupinosis

Chronic lupinosis is caused in sheep by the presence in lupin species of the fungus *Phomopsis leptostromiformis*, causing liver damage. The condition has not been reported for horses, in which acute lupin poisoning may occasionally occur (see 'Toxic legumes and other species', this chapter).

Plant toxins

In the plant world there is a vast array of substances that are toxic and hazardous to horses when consumed in disproportionate amounts. Many of these are known as alkaloids, which are organic, basic compounds, although the chemical diversity of all hazardous compounds is considerable, and their range of toxicity to the horse is wide. Numerous broad-leaved plant species on or around pastures are toxic to a

greater or lesser degree as a consequence of their production of endogenous toxins. Access by horses to shrubs, trees and hedgerow plants is a typical cause of intoxication, although minor intoxication can occur, for example, from widely abundant members of the Ranunculaceae. In the author's experience, heavy infestation of pastures by some members of this family causes buccal irritation in horses. Members of this family contain an irritant yellow volatile oil, protoanemonin, but in differing amounts. This substance can cause irritation to the mouth of grazing animals, but hay containing Ranunculaceae is safe, as far as *this* chemical is concerned, as the curing process causes precipitation of protoanemonin in a harmless form.

More severe toxicosis is caused by contamination of upland hay by bracken which causes progressive ataxia. The whole plant is toxic and the principles include a cyanogenetic glycoside in relatively low concentrations, a thiaminase (causing the ataxia), an aplastic anaemia factor, a factor causing haematuria and a carcinogen, although these last three may be identical. Water meadows are another source of hazardous plants and dry parched grassland, on which drought-resistant harmful shrubs survive, is yet another. In many cases conclusive diagnoses are unlikely.

Photosensitization

Photosensitization refers to the production of skin lesions caused by the interaction of sunlight with exogenous substances that are capable of activation by solar radiation to form free radicals. Primary photosensitization is caused by sunlight reacting directly with dietary substances in the skin after absorption. Examples include hypericin contained in St John's worts (*Hypericum* spp. especially *H. perforatum*), fagopyrin in buckwheat (*Fagopyrum esculentum*) and toxins in bog asphodel (*Narthecium ossifragum*), in species of *Vicia* and sometimes in lucerne (*Medicago sativa*), alsike (*Trifolium hybridum*) and red clovers. On other occasions, therapeutic drugs may be incriminated. Lucerne photosensitization is caused by pheophorbide- α contained in the leaves of dried lucerne. The pheophorbide- α is formed by breakdown of chlorophyll under the influence of chlorophyllase, during processing. There is a higher activity of this enzyme in legume forages than in grass.

Many absorbed toxins are detoxified in the liver and excreted in bile. This process is obviously less efficient where there is a measure of liver dysfunction. Secondary photosensitization occurs when a damaged liver is unable to clear, for example, phylloerythrin, a photodynamic, chlorophyll metabolite, in the bile. Skin reactions are most likely to be observed following the consumption of large amounts of green forage by horses that have received some hepatotoxic agent. Numerous warm-season grass species cause secondary photosensitization, characterized by photophobia and severe dermatitis. These species include *Panicum* and *Brachiaria* which contain steroidal saponins, causing liver damage, possibly by interaction with mycotoxins.

Facial eczema in sheep is secondary photosensitization caused by the mycotoxin sporidesmin, contained in spores produced by the fungus *Pithomyces chartarum*. The toxin causes free-radical liver damage. As Cu strongly catalyses the oxidation, protection is afforded by Zn supplementation that reduces Cu absorption and blood Cu level. Zn also binds with a sporidesmin metabolite, preventing its autoxidation. It is of concern in New Zealand, where sheep and cattle develop severe dermatitis of light-skinned areas of the body.

Toxic legumes and other species

Yew (*Taxus baccata*) is the most toxic plant (non-legume) in the UK, and little more than 100g of it will kill a horse by cardiac arrest. The second most poisonous is laburnum (*Laburnum*), a member of the legume family, which contains many plants known to cause damage to horses, among them broom (*Cytisus scoparius*) and lupins (*Lupinus*). The toxicity of lupins is principally confined to the seeds, and the various strains differ in their potency. Sweet lupins (*L. lutens*) have a low alkaloid content and are grown on poor land as a source of fodder. If horses eat them death is rare and is caused by respiratory paralysis, not by liver damage. An accumulative poisoning associated with progressive liver damage (chronic lupinosis) occurs in sheep and horses in Australia. Here the causative agent is a fungus growing on the lupins.

Sweet clover or melilot (*Melilotus*) contains coumarin which is broken down to dicoumarol in hay made under bad harvesting conditions, or during moulding. Dicoumarol prolongs blood clotting time. Both white and red clovers may contain toxic factors. Both species contain appreciable amounts of oestrogens which are also found (in much higher concentrations) in subterranean clover (*Trifolium subterraneum*) grown on light soils. Some reports indicate the presence of oestrogenic activity in moulds infecting the clover leaves. These hormone-like substances have been associated with infertility and increased teat length of sheep and it is an open question as to whether any comparable problem occurs in grazing mares.

Alsike clover (*Trifolium hybridum*, derived from Alsike, a village near Uppsala, Sweden, but the clover is not a hybrid), a tall-growing plant with pinkish white flowers, may cause poisoning in horses. Wright *et al.* (2003a) observed inappetence, lethargy, dehydration, pyrexia, oedema of the lower limbs, hepatitis, icterus, diarrhoea and petechial haemorrhages and ulcers of the oral cavity when adult TBs received a diet of sweetfeed, soaked sugar-beet pulp and hay containing alsike clover in amounts exceeding 20% in some bales. Two genera of vetches in the USA, milk vetches or locoweeds (*Astragalus*) and the related *Oxytropis* (see Table 10.17), are implicated in several abnormal conditions of horses. One caused by locoweeds is recognized as eliciting irreversible nervous signs.

Plants of the genera *Swainsona*, *Oxytropis*, *Astragalus* and *Ipomoea* cause α -mannosidosis, a lysosomal storage disease of herbivores. Loretto *et al.* (2003), in southern Brazil, investigated this disease in ponies. It is characterised by stiff gait,

muscle tremor, abdominal pain and death, following introduction to pastures heavily infested with *Sida carpinifolia*. Multiple cytoplasmic vacuoles in swollen neurones of the cerebellum, spinal cord and autonomic ganglia were noted *post mortem*. There was also marked vacuolation of the renal proximal convoluted tubular cells. Lectin histochemistry of neuronal vacuoles revealed staining that coincided with that of inherited mannosidosis.

Spotted locoweed (*Astragalus lentiginosus* var. *diphysus*), found in pinyon–juniper woodlands on the Colorado Plateau is readily eaten by horses, and the toxin it contains, swainsonine, causes anorexia and behavioural instability (Pfister *et al.* 2003). Another locoweed (*Oxytropis sericea*) also causes a serious problem on rangelands in the western USA. Pfister *et al.* (2002) demonstrated that lithium chloride, administered by stomach tube at a rate of 190 mg/kg BW, conditioned a strong and persistent aversion to this weed, although some horses may require more than one pairing of the aversion agent with the taste of the weed for the effect to persist.

Although none of the poisonous members of these genera are found in the UK, certain pasture species of *Vicia* and *Lathyrus* found there are mildly toxic. In parts of the USA, locoweeds begin their growth in late summer and remain green through the winter. They must be grazed for a period before poisoning is obvious. Some species are Se accumulators, containing up to 300 mg/kg, and are poisonous for this reason.

Grass toxins

Toxins are rarely produced by grass tissues, although several species are known to do so.

Kikuyu grass poisoning

In addition to its soluble oxalates, Kikuyu grass (*Pennisetum clandestinum*) causes a poisoning of horses that is probably related to saponins that accumulate during periods of rapid growth of the grass. Signs include false simulated drinking, anorexia, depression, pilo-erection, drooling, colic, grinding of teeth, cessation of intestinal movement and cessation of defecation.

Reed canary grass poisoning

Reed canary grass (*Phalaris arundinacea*) is a forage grown on wet, poorly drained soils. It contains at least eight different alkaloids that have caused poisoning in farm livestock. Reed canary grass is relatively unpalatable and this may be one reason why there have been no reported cases of intoxication in horses.

Oxalate poisoning

Various tropical grass species, including buffel grass (*Cenchrus ciliaris*), pangola grass (*Digitaria decumbens*), setaria (*Setaria sphacelata*) and Kikuyu grass (*Pennisetum clandestinum*) contain soluble oxalates that react with Ca to form

insoluble Ca oxalate, reducing Ca absorption. This causes mobilization of bone mineral and secondary hyperparathyroidism, or osteodystrophy fibrosa in horses. Cattle and sheep are less affected, but not unaffected, owing to degradation of oxalates in the rumen. Concentrations of 5g or more soluble oxalates/kg DM in forage grasses induce the condition in horses. Oxalate content of these grasses is highest under conditions of rapid growth. Oxalate toxicity is also discussed under 'Tropical grassland and forages', this chapter.

Poisonous trees

Many tree species contain substances that are toxic to a greater or lesser degree (Table 10.17). The toxins may be present in the leaves, bark and/or fruits. A high proportion of horses bedded on black walnut (*Juglans nigra*) wood shavings develop laminitis. An aqueous extract of the heartwood given by stomach tube was shown to cause limb oedema, mild sedation and Obel grade 3 or 4 laminitis (see Chapter 11) within 12 hours. The signs are inconsistent with those caused by carbohydrate overload. The toxin has not been identified.

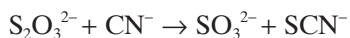
Cyanogenic species

Hydrogen cyanide toxicity

Cyanide toxicosis is caused by the inhibition of cytochrome oxidase (EC 1.9.3.1), a terminal respiratory enzyme in all cells, depriving the cell of ATP. Thus, signs of acute intoxication include laboured breathing, excitement, gasping, staggering, convulsions, paralysis and death. In tropical and subtropical countries, enzootic equine cystitis and ataxia occur in horses grazing fresh summer annual forages of the genus *Sorghum* (Johnson grass, Sudan grass, *S. sudanense* and common sorghum). However, acute toxicosis is more likely with the consumption of sorghum hay, or especially with ground and pelleted sorghum hay, owing to the rapid rate of intake and of cyanide release. Ensiling markedly reduces the risk of cyanide toxicosis. Sorghums contain a cyanogenic glycoside, dhurrin, from which free cyanide can be released by enzymatic action. The glycoside and the enzyme are contained in different plant cells, but damage to the plant from wilting, trampling, frost and drought result in the breakdown of cell walls, with the mixing of the juices and release of free cyanide. Arrow grass (*Triglochin*), wild black cherry (*Prunus serotina*), choke cherry (*P. virginiana*), pincherry (*P. pennsylvanica*) and flax (*Linum*) also contain cyanogenic glycosides which are readily hydrolysed to hydrogen cyanide. They are most toxic during rapid growth immediately after a freeze. Heavy N fertilization, wilting, trampling and plant diseases may increase the hazard. Silage or haylage produced from the grasses is risky. Cystitis, or inflammation of the urinary tract, and incontinence are more common in mares than in stallions or geldings, but posterior ataxia, from which they seldom recover, is manifested in all horses.

Thiocyanate

Cyanide is readily detoxified in animal tissues, during low rates of intake, by reacting with thiosulphate to form thiocyanate, the blood and urinary concentrations of which increase during chronic exposure:



However, the chronic production of thiocyanate (SCN^-) can induce ataxia, degenerative lesions of the central nervous system, goitre and a deficiency of S, owing to urinary loss of S as thiocyanate.

Hepatotoxins

Malnutrition can increase the risks of many toxins, or one toxin may compound the effects of another. Although definite conclusions are usually unlikely there are several well-known interactions in the field. *Heliotropium europaeum* (Boraginaceae) is a shrub that produces the pyrrolizidine alkaloid hepatotoxins heliotrine and lasiocarpine, causing lesions, associated with Cu accumulation and a subsequent haemolytic crisis of chronic Cu poisoning. *Heliotropium europaeum* would not normally be grazed if there was other more appetizing vegetation. A straightforward case was presented to the author once, in which severe liver damage and death occurred among sheep. These animals browsed in an environment where the pink flowers and green foliage of *H. europaeum* were about the only species not desiccated and these plants were set against the background of a blue streak of Cu salts in the bed of a dried-up wadi.

Seneciosis

In the UK and many temperate lands the most common source of pyrrolizidine alkaloids is ragwort (*Senecio jacobaea*), frequently consumed in hay. Seneciosis is a disease caused by the ingestion of certain plants of the genus *Senecio* (Compositae) which induce liver enlargement, degeneration, necrosis, cirrhosis and ascites. A constant feature has been occlusion of the centrilobular vein (veno-occlusive disease – VOD), briefly reviewed by Hill (1959). Although ragwort may be a hazardous member of this group, the inconspicuous groundsel (*S. vulgaris*) is a source, albeit a lesser one, of the toxins. There have been many reports of seneciosis from the USA, Europe and other continents. The worldwide distribution of the disease is attested by the variety of names used to describe it: ‘walking disease’ of horses and cattle in Nebraska, ‘walking about’ disease of horses and cattle in Australia, ‘Pictou’ disease in Nova Scotia, ‘Zdar’ disease in former Czechoslovakia, ‘Schweinberger’ disease in Germany, ‘Dunziekte’ of horses and cattle in South Africa, and ‘Winton’ disease in New Zealand. It is also recognized in goats, chickens, quail, doves and pigs. *Crotalaria* (Leguminosae) and *Heliotropium* have similar hepatotoxic properties so their effects are included under the umbrella term seneciosis.

Dry summers leave many paddocks and pastures in a poor state, with typical grass

species dying back. On the other hand, deep-rooted and drought-resistant weeds survive. In this weather, ragwort and its seeds spread and an increase in the frequency of liver damage among horses and other herbivores can be expected to occur. The different resistances of grazing animals to this toxin reflect a variation in the efficiency of its urinary elimination (Holton *et al.* 1983). Elevated plasma concentrations of GGT (EC 2.3.2.2) are a useful early indicator of the hepatic damage caused by ragwort toxins. Horses with ragwort-damaged livers should be given a well-balanced diet containing good-quality protein supplemented with B vitamins and trace elements.

The common comfrey (*Symphytum officinale*) contains at least nine potentially hepatotoxic pyrrolizidine alkaloids in its leaves and roots, which are less toxic than those in ragwort. Comfrey has been recommended for inclusion in horse feeds, but this recommendation cannot be supported, owing to the risk of liver damage.

Aflatoxicosis

Liver damage through aflatoxicosis, derived from *Aspergillus flavus* intoxication, to which the horse is very susceptible, is less likely now within the EU as a result of legislation. This toxin was first described in groundnuts, or peanuts (*Arachis hypogaea*), but has subsequently been detected at lower concentrations in other plant species, including some cereal grains.

HOMEOPATHY

The principle of homeopathy is to give a potentially toxic chemical, which in large doses causes the signs of a specific disease, but which in small doses is said to cure that disease. Unfortunately, much of the evidence supporting the claims is anecdotal and there is a need for these claims to be subjected to acceptable experimental methods of examination. The remedies used in homeopathy are extracted from materials in the animal and plant kingdoms and from natural minerals. These include:

- bryonia (wild hops);
- belladonna (deadly nightshade);
- silica (pure flint);
- sulphur;
- sepia (Cuttlefish ink); and
- apis (honey bee) (Evans 1995).

A potential development of some treatment procedures is hazardous, e.g. 'nosodes'. Several of the treatments involve use of irritant substances extracted from plants, e.g. pulsatilla from *Pulsatilla nigricans* (Ranunculaceae), arnica from flowers of *Arnica montana* and related Compositae, with well-known pharmacological properties. The general question is whether the very low doses typically used have any measurable effect, whereas high doses could be harmful.

STUDY QUESTIONS

- (1) What would you propose should be done about worn-out, worm-infested pastures on (a) heavy clay and (b) light alkaline soils?
- (2) Where (a) sheep or (b) cattle can be purchased, how would you propose a mixed pasture management system should be organized for a 50-ha stud with 30 mares and followers?
- (3) What would be the sequence of decisions in planning to make haylage for a stud of 20 mares?

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Chapter 11

Pests and Ailments Related to Grazing Area, Diet and Housing

For a surfeited horse. Take a handful of pennyroyal, half a handful of hyssop, an handful of sage, an handful of elder leaves or buds, an handful of nettle tops, fix large sprigs of rue, and handful of celendine, cut small and boiled in three pints of stale beer, which must be boiled to a quart.

Sir Paulet St John 1780

ARTHROPOD PARASITES

Lice

There are two species of horse lice: *Haematopinus asini* is a blood sucker and *Damalinia equi* lives on skin scales. The females lay eggs on the hair and a greater problem of scratching or rubbing is frequently observed in the winter than in the summer so that many are lost when the winter coat is shed. Control is achieved by dipping, spraying or dusting with insecticides, but a second treatment should be undertaken to kill those that will hatch from eggs already laid.

Ticks

Grazing horses, particularly those sharing ground with wild grazing and browsing animals, are prone to infestation by ticks, which can transmit diseases. However, the cattle and sheep tick in the UK does not usually cause symptoms in horses. In the USA the soft tick (*Otobius meгинi*) lives deep in the ears, but the adults, which do not feed, live in cracks in stables, fences and under troughs where they also lay eggs. Larvae of hard ticks (*Dermacentor andersoni*; *Amblyomma americanum*) are found in various places on the horse, and the adults, after mating, fall to the ground, where eggs are laid in secluded locations. The tropical horse tick (*Dermacentor nitens*), whose primary host is the horse, transmits equine piroplasmosis, a protozoan blood disease. Insecticide should be applied to all parts of the skin where the ticks may be attached, including the ears. As the parasites spend long periods off the host, the grass and other areas around the stable should also be treated. With slight infestations, the ticks can be detached by application of chloroform to release their mouthparts.

Lyme disease was first described in 1977, following an outbreak in man of arthritis in Lyme, Connecticut. The disease is caused by *Borrelia burgdorferi* and antibodies to this spirochaete bacterium have been detected in the sera and synovial fluid of

horses in the UK (there is, unfortunately, a lack of specificity owing to cross-reacting antibodies), the majority of which did not display clinical manifestations of Lyme disease. These signs include arthritis, myositis, weight loss, fever, laminitis and possibly meningoencephalitis. Diagnosis requires histological demonstration of silver-stained spirochaetes in skin biopsy specimens, or the (difficult) culture of the organism from blood or cerebrospinal fluid. The bacterium is transmitted by several species of the ixodid tick (*Ixodides ricinus* and *I. persulcatus* in Europe) in the northern hemisphere, which feed on many species of animal. The susceptibility of these animals to infection with *B. burgdorferi* is largely unknown, although they undoubtedly affect the prevalence of the infection. Acute infection responds to appropriate antibiotic treatment, but the chronic arthritis is often unresponsive.

Mites

Mites (*Psoroptes equi*, *P. cuniculi*, *Chorioptes bovis*) cause itch or scabs and may be controlled by dipping or spraying with insecticide. As a general rule, high-pressure sprays frighten horses so that low-pressure hand-pumped sprayers are preferable and the horse should be confined to a chute during treatment.

Biting midges

An intensely pruritic dermatitis (Sweet itch), which occurs during the summer months and is quite common in Ireland, is probably caused by species of *Culicoides* (Baker & Quinn 1978), a blood-sucking midge whose saliva induces an immediate-type hypersensitivity. Plasma zinc has a potent immunomodulatory capacity, influencing T-helper-cell organisation and cytokine secretion. Stark *et al.* (2001) demonstrated a negative association between plasma zinc concentration and the severity of *Culicoides* hypersensitivity. Whether this relationship is an evoked response, or part of the cause needs investigation. Stock should not be grazed over wet areas where the midges are found and they should be stabled before dusk. Some control is achieved with antihistamines.

Flies

Several species of fly are more of a nuisance than a direct cause of trouble. The warble fly (*Hypoderma lineatum*) can cause some damage, mainly in young horses when the larva penetrates the skin of the legs and wanders under the skin to the back. When it is nearly ready to emerge it should be poulticed. The screw-worm fly (*Callitroga hominivorax*) does not occur in Western Europe and has probably been eliminated from the USA. It causes wounds in the skin in which it lays eggs from which the larvae hatch. Direct treatment of the wounds with insecticide is appropriate. Several species of botfly (*Gastrophilus*) are widespread. The adult lays eggs on

the breast and around the mouth and gums of the horse. The larvae are swallowed and attach by hooks to the stomach or small intestine, detaching when fully grown and pupating in the manure. Some control can be achieved by sponging areas of the skin on which eggs are attached to hair, using water at a minimum of 49°C (120°F). Where necessary, ivermectin or another insecticidal anthelmintic can be given orally.

General stable hygiene is a major factor in the control of all flies, including the immediate removal of dung, contaminated feed and bedding. Routine grooming may assist not only by removing potential trouble but by ensuring that there is a regular scrutiny of the horse's coat.

Blister beetles

Blister beetles (*Epicauta* spp. and *Macrobasis* spp.) are lethal when ingested by livestock. Various species are distributed throughout Canada and the USA. They range from 0.8 to 2.7 cm in length and they may be black, black with grey hairs, black with red or yellow contrasting stripes, yellow with black stripes, metallic green or purple. They travel in swarms and feed on flowering plants such as lucerne or clover. When lucerne hay is harvested, the insects can be crushed and incorporated in the bale. Upon ingestion, cantharidin, an extremely stable toxin, for which there is no known remedy, is released. It is claimed that 6 g of the beetle are sufficient to kill a horse. Cantharidin causes severe inflammation of the oesophagus, stomach and intestines and during urinary excretion causes severe irritation to the urinary tract. The horse develops colic and dies within 48 hours.

Hay baled in July and August is more likely to be infested than that cut earlier. If the insects are detected, they may be present in considerable numbers as a consequence of their swarming nature. By knocking biscuits of hay before feeding at least one or two may fall out from infested material. The existence of the risk is, however, not a justification for the exclusion of lucerne hay from the diet.

WORM INFESTATIONS

In temperate countries, helminth infection (helminthiasis) in horses is limited to GI nematodes, including lung worm, and to liver fluke (trematodes). In tropical countries, however, horses suffer spirurid and filarial infections as well. Foals may be heavily infected with migrating large strongyle nematode larvae with a pre-patent period of 6–12 months and therefore injurious infections can be present for many months before eggs are detected. Adult horses may become severely parasitized by migrating larvae, even if wormed, when sharing pastures with horses that are not wormed.

The determination of the severity of worm infestation is no simple matter. Faecal egg counts simply reflect the presence of egg-laying worms. The only reliable means of establishing the degree of parasitism by GI nematodes is to analyse serum

proteins. Alpha and beta globulins peak in concentration six months after infection and thereafter the latter of these proteins declines. There is a coincidental depression in serum albumin and eventually in haemoglobin. Worm egg counts are, however, of use in assessing anthelmintic efficacy in control schemes. Raised eosinophil counts reflect only migrating larvae so that these counts may not differ between treated and untreated animals. The counts tend to be highest in July and August in the northern hemisphere and so are not diagnostic. Both small and large infections with strongyles cause an elevation in the immunoglobulin IgG (T) concomitantly with depressed serum albumin before a patent infection occurs.

GI parasitic nematodes

The eggs of parasitic nematodes such as *strongylus* spp. pass out in faeces from adults in the gut and hatch into the first and second larval stages while contained in droppings on pasture. These stages are not infectious [some species undergo maturation through all three stages within the egg, e.g. ascarids (*Parascaris equorum*), and so are more resistant to deleterious environments, and may survive for years]. Larvae of the third larval stage of *Strongylus* move out onto the surrounding blades of grass and are infectious. These developments require moisture. Once eaten, larvae of the third stage pass through two more stages before becoming adult, mating and beginning to lay eggs. The complete cycle takes about eight weeks in the summer, the rate depending on the ambient temperature. As autumn approaches, an increasing number of the parasites stop development and hibernate in the gut wall, to emerge again in the spring. The larvae of some nematode species migrate within the body, passing along blood vessels through the liver and lungs, causing damage. Larvae in the pasture can survive winter to infect horses the following spring. Most parasites are 'host specific', that is, parasites of sheep and cattle will not generally infect horses, but those of donkeys will infect horses.

***Strongyloides westeri* (threadworm)**

Threadworms are very small worms that live in the small intestines of foals. Foals are infected shortly after birth, either by ingestion of colostrum and milk containing the larvae, or by the larvae penetrating the skin. Heavy infestations cause sufficient damage to the intestinal lining to precipitate diarrhoea, loss of appetite and dullness. Foals are susceptible up to six months of age, after which, they will usually, have developed immunity.

***Parascaris equorum* (large roundworm)**

Large roundworms can reach a length of 50cm and their lifecycle is 10–12 weeks. Eggs containing infective larvae are picked up from the pasture, or from contaminated stable bedding. The larvae migrate through the vascular system to the liver and lungs, before returning to the small intestine, where they develop into adults

and lay eggs. Adult worms arrest growth and harm the appearance of foals. Heavy infestations can block the gut and migrating stages in the lungs cause 'summer colds' with fever, coughing and loss of appetite.

***Cyathostome* spp. (small redworm)**

Small redworms are major parasites. They are very small and live in the large intestine. The life cycle is 5–18 weeks and infective larvae are eaten with the pasture during the summer. The larvae of these worms may be predominant in pastures in the UK during the summer. The seasonal emergence of huge numbers of larvae from the gut mucosa during the late winter and early spring can cause debilitating acute diarrhoea, weight loss, colic and even death. They may be the most common cause of diarrhoea among adult horses in the UK. Horses infected with larval cyathostomiasis, or *Strongylus* spp., frequently show elevated serum β -1-globulin concentrations. Inflammation caused by cyathostomes can also elevate α -2-globulin; and leakage from the gut causes depressed serum albumin levels (protein-losing enteropathy). These facts may be helpful in diagnosis of horses that have recently received anthelmintic treatment, and which may not be passing larvae. Most anthelmintic treatment will not affect larvae already encysted in the mucosa. A five-day treatment with fenbendazole has shown efficacy, but benzimidazole- (fenbendazole) resistant cyathostomes have been detected in half the horses sampled in central Scotland by Chandler & Love (2002). Oral moxidectin, however, effectively suppressed faecal cyathostome egg output over a sustained period. Ivermectin treatment of mature horses infected with cyathostomins resistant to fenbendazole and pyrantel was similarly effective (Little *et al.* 2003).

Apart from this, improved biological control should be considered. The nematode-trapping microfungus, *Duddingtonia flagrans*, is an aggressive trapper of free-living stages of nematode parasites. Fernández *et al.* (1999) demonstrated that this nematophagous fungus, included at the rate of 1×10^6 spores/kg BW in a feed supplement, led to >90% reduction in faecal pat contamination with horse strongyle third stage larvae, without any adverse effect on the supplement. Waller (1999) considers that the biological approach should be used in conjunction with anthelmintics, pasture hygiene and grazing management.

A good-quality dietary protein will help compensate for blood albumin loss.

***Strongylus* spp. (large redworm)**

Large redworms are reddish-brown worms, 2–5 cm long and have a life cycle of 6–11 months. The larvae have been detected in smaller numbers on pastures in the UK in recent years. Picked up from the pasture, the larvae of *Strongylus vulgaris* penetrate the gut wall and migrate to the main coeliac artery, where they are responsible for severe damage and blood clots. These thrombi can become dislodged and then block smaller branches of the artery. This interruption to blood supply typically causes colic.

Cyathostomes and large redworms are an important cause of debility in young grazing horses. In Denmark, Thamsborg *et al.* (1998) subjected foals of three to six months of age to pastures with either low, or high, larval contamination by large strongyles and cyathostomes for four weeks in September. The high level of contamination caused debility, inappetence and intermittent diarrhoea without colic. A transient neutrophilia and eosinophilia occurred two to eight weeks after the start of exposure, followed later by anaemia and decreased serum albumin. A marked hyperbetaglobulinaemia was detected at 16–20 weeks after the start.

***Oxyuris equi* (pinworm)**

Pinworm females are up to 10 cm in length. The life cycle is 4–5 months. The adults migrate to, and lay eggs on, the skin surrounding the anus, causing irritation. Rubbing of the anal region causes the opening of wounds, the hair to be removed and the eggs to drop off to the stabling and pasture, from which they are picked up.

***Dictyocaulus arnfieldi* (lungworm)**

The life cycle of lungworm is 2–4 months. Infective larvae are picked up from the pasture. The larvae are swallowed and migrate through the bloodstream to the lungs, where they develop into adults. Eggs are laid there, coughed up, swallowed and passed out in the faeces. Although donkeys frequently act as carriers, they and foals rarely show signs. The examination of faeces for larvae is useful in detecting carrier animals responsible for spreading the infection. Most horses possess some resistance and do not develop patent infections. However, where they do, the pre-patent period before larvae may be found in the faeces is three months. The larvae may remain in a state of retarded development and hosts can elicit persistent coughing for periods exceeding one year in adults, during which time resort to veterinary lavage of the trachea for the detection of larvae in the washings is a rational diagnostic technique, even though the larvae are not readily demonstrated. Although lungworm evoke eosinophilia there is no detectable change in serum proteins. Management of lungworm infection requires identification of the carrier, which may be a donkey, or unhealthy mare shedding faecal larvae without showing signs. This animal should then be removed and treated with effective anthelmintics. Fluke and lungworm are not as economically significant among horses in the UK as are GI nematodes.

***Trichostrongylus axei* (stomach hairworm)**

Stomach hairworms have a life cycle of three weeks, they live in the stomach causing damage and irritation and they are able also to infect cattle and sheep. Larvae of this parasite have been found to assume major importance on pastures in the UK from August until October.

***Habronema muscae* (large-mouthed stomach worm)**

The adults of the large-mouthed stomach worm live in the stomach. Eggs are passed out in the dung, where they hatch and are picked up by fly maggots feeding in the dung. The larvae are carried in the mouthparts of the fly. As the fly feeds it passes the larvae to the feeding horse, which swallows the larvae. Larvae deposited on sores and wounds of the horse's skin do not complete their life cycle, but cause intense irritation and 'summer sores'.

***Onchocerca* spp. (neck threadworm)**

Adults of the neck threadworm live in tendons and ligaments. The larvae (microfilariae) live under the skin and in eye tissue, and are taken up by feeding midges. Microfilariae in the eyes cause problems.

Other worm parasites***Gastrophilus* (bot fly)**

The bot fly lays eggs on the legs and face of the horse. These hatch and enter the mouth, where the larvae live in the tissues of the lining and the tongue for several weeks before entering the stomach. The larvae attach to the stomach wall, where they remain until the following spring. These larvae can cause ulceration and perforation of the stomach wall during this time. They then pass out in the faeces. The larvae mine underground and pupate. The adult flies emerge during the summer to lay eggs. The lifecycle is one year.

***Anoplocephala perfoliata* (tapeworm)**

Horses with obstructions of the ileum and caecum are frequently found to harbour the tapeworm, and horses with concurrent infestation run an increased risk of ileo-caecal colic (Proudman & Edwards 1993). Proudman *et al.* (1998) concluded from a survey that many of their spasmodic colic cases, and most ileal impaction cases, were associated with tapeworm (*A. perfoliata*) infection. Tapeworm eggs are passed out, contained in proglottides (segments), in the dung. The eggs are consumed by free-living oribatid mites, which are, in turn, eaten with the grass by the horse. The adult worms attach to the wall of the intestines at the junction of the small and large gut.

Control of GI worm parasites

Control requires an effective worming programme and good pasture management. The essence of control is to reduce the number of infective larvae on pastures grazed by susceptible stock, particularly those under three years old. There is some evidence that tolerance to both strongyle and ascarid nematodes is developed, so that stock should not be kept entirely isolated from infective sources. Faecal egg counts

Table 11.1 Treatment programme for GI parasites in northern latitudes.

	Treatment	Purpose	Additional activity
January			
February	C for 5 days	Encysted small redworm	
March	B double dose	Tapeworm	Faecal examination of all stock
April	A, B or C every 6–10 weeks	Grazing season	
May			
June			Faecal examination of early foals
July			
August			Faecal examination of late foals
September	B double dose		
October	A or C	Mid-October to end of December migrating large redworm	Faecal examination of all stock
November	C	Late October to end of December encysted small redworm	
December	A	Bot	

A, avermectins; B, pyrimidines; C, benzimidazoles.

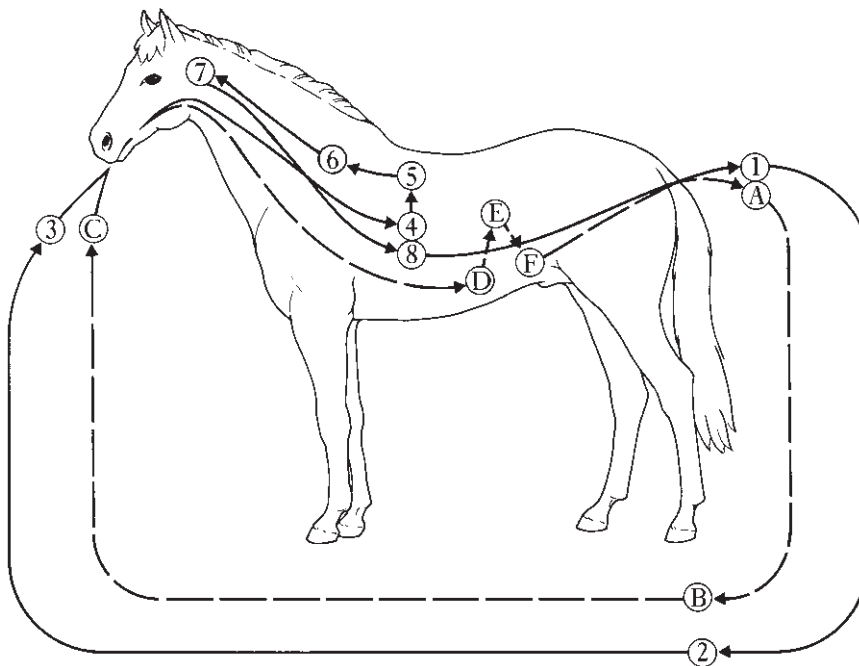
reflect only the activity of adult worms in the intestines and may not give a good indication of the seriousness of a strongyle infection.

Horses should be treated orally with anthelmintics on arrival at a stable or as directed by the veterinary surgeon; however, an initial dose, much larger than normal, of an effective wormer may be prudent in cases of severe infection with strongyles as such doses of thiabendazole, or fenbendazole, can be larvicidal. Veterinary guidance is essential. Moderate but closely defined doses of oxfendazole or ivermectin (Dunsmore 1985) have been shown to possess efficacy against adult ascarids and small and large strongyles at all stages from egg to adults, including migrating larvae. Ivermectin also controls bot-fly larvae.

Young stock should always have access to the cleanest pasture until they have developed some tolerance to worms. Mares must therefore be properly treated so that they do not pass on any severe infection to their offspring; droppings can be removed expeditiously by the use of vacuum cleaner attachments to tractors. Stabled horses should also be treated routinely, particularly where they have been given access to pasture, even for short periods, in the summer. Table 11.1 gives a simple routine of treatment. The life histories of two species are described in Fig. 11.1.

Strongyles (redworm)

Strongyle eggs develop into infective larvae only in the period between March and October, especially in warm weather. Infective larvae can survive the winter in the

**Ascarids***Parascaris equorum*

(in young stock)

- (1) Immature eggs appear in droppings from 12–15 weeks after infection
- (2) Eggs may become infective in pasture or stable within 2 weeks, or remain dormant for up to several years
- (3) Mature eggs ingested by susceptible foal or yearling
- (4) Second stage larva hatches from egg and penetrates intestinal wall
- (5) Larva reaches liver from 7 days after ingestion of infective egg
- (6) Larva reaches lungs from 7–14 days after ingestion
- (7) Larva coughed up and swallowed
- (8) From fourth to approximately thirteenth week larva grows and matures in small intestine and begins shedding eggs 12–15 weeks after infection

Strongyles*Strongylus vulgaris*

(in young and adult stock)

- (A) Non-infective eggs shed on pasture
- (B) Larva matures within egg case on the pasture during the grazing season
- (C) Third-stage infective larva ingested
- (D) Larva penetrates submucosa of intestine within a few days
- (E) By 14 days after ingestion larva has reached the anterior mesenteric artery where it develops over a 4-month period.

Fig. 11.1 Life history of a roundworm (*Parascaris equorum*) and large redworm (*Strongylus vulgaris*).

UK, but in the spring there is a rapid disappearance of these larvae from pasture with increasing ambient temperature. Overwintered larvae die out by June. Nevertheless, in the early grazing season this source augments that from eggs passed by other horses throughout early grazing. The high level of infectivity accumulating

during the summer on pasture can be contained by regular anthelmintic dosing at four-six-week intervals, which complements the management procedures given in Chapter 10. For strongyle control there is little point in dosing foals less than two months old as the pre-patent period of small strongyles is 8–10 weeks and the developmental stages are not susceptible to most anthelmintics in the normal dose range. Badly infested pastures may need ploughing and reseeded, or at least should be rested till June, by which time overwintered larvae will have largely gone. However, young stock should not be given access to them until July or August before which time grazing should be restricted to cattle or sheep.

Ascarids (large roundworms)

Ascarid infection is common in horses under three years old, by which time a considerable measure of resistance will have developed. Foals are especially susceptible and it is thought that nearly all become infected without necessarily developing signs, owing to anthelmintic control measures and increasing immunity. The migrating larvae damage successively the liver and lungs within 14 days of infection. Eggs occur in the faeces from 80 days of age. Eggs acquired by the young foal through coprophagia of the dam's droppings are normally immature and pass passively through the foal's intestines.

Clinical signs of severe infection include pyrexia, coughing, nasal discharge, nervousness, colic and unthriftiness. To preclude this, foals should be treated at four-week intervals from one month of age for the control of the intestinal stages. Eggs can remain viable on pasture over winter and in suitable conditions some may persist in the environment for many years. The pasture management advised for strongyles is also applicable to the control of ascarids. For a more detailed discussion of parasitic worm control in the stud the reader is referred to Rosedale & Ricketts (1980).

Various wormers have a different spectrum of species against which they are effective, differences in activity against adults and larvae and differences in the number and frequency of dosing. Inadequate dosing can lead to the development of resistance to wormers, and small redworms are now widely resistant to the benzimidazoles. Three families of wormers are used:

- (1) avermectins;
- (2) pyrimidines;
- (3) benzimidazoles.

There are several principles to worming:

- Know the weight of each horse so that correct dosages may be given.
- Treat any horse on arrival at a new establishment and keep it stabled separately for at least two days.
- Treat all horses grazing together at the same time and with the same product. If they are at pasture during the winter continue treatment during that period.
- Keep a diary of the date and product used for each horse.

- Do not overstock paddocks.
- Worm horses two days before moving them to clean pastures.
- Collect dung from the paddocks two to three times per week. This is probably *the most important procedure* in parasitic worm control. Moreover, strict adherence to this procedure can increase the grazing area by 50%, by eliminating the characteristic separation of horse pasture into roughs and lawns.
- To rest a pasture, do not graze it with horses from autumn until the following mid-summer. Where possible use cattle or sheep to clear infected pastures of parasites.
- Rotate wormers used in the grazing season on an annual basis, not every time the horses are wormed. The rotation should be based on changing from *one to another* of the three chemical groups listed above.

PROTOZOAN PARASITES

Ingestion of faeces-contaminated food or water can lead to transmission of the protozoa *Giardia duodenalis* and *Cryptosporidium parvum*, in locations where these organisms exist. In the Sierra Nevada Range of California, Atwill *et al.* (2000) detected *G. duodenalis* in the faeces of 4.6% of packstock horses and mules and an estimated prevalence for *C. parvum* of <2.4%. Suspect water sources should be fenced off if possible.

LIVER FLUKE

Reference to the infectivity of liver flukes (*Fasciola hepatica*) in horses was made in Chapter 10. Their presence may be detected by faecal egg counts and their influence by liver-function tests, indicating liver damage. Untreated cattle and sheep encourage their spread, and snails are an obligatory intermediate host.

AILMENTS RELATED TO DIET

Microbial spoilage of feeds

(See also 'Feed storage', Chapter 5.) Bacteria grow in feeds where the moisture content is over about 16%. This may result from poor drying or from secondary water uptake in humid atmospheres and condensation on the surface of feeds. Cereal grains that have been ground or crushed, or feeds, such as bran, with a large surface area, are more susceptible to bacterial and mould growth.

Endotoxaemia and laminitis

Evidence shows that endotoxaemia and laminitis are related in the horse. The dietary causes of acute laminitis and endotoxaemia are, to a considerable extent,

limited to the consequences of an excessive consumption of readily fermentable carbohydrate by horses that have been inadequately adapted to the diet. However, equine laminitis is a local Shwartzman-type hypersensitivity reaction which may be provoked by several other agents that are neither antigenic nor dietary. These could include endotoxin.

Sensitization is a predetermining factor, so a history of laminitis, the endogenous release of corticosteroids in response to stress, previous exposure of tissues to endotoxin, followed by stress, are factors that increase the probability of laminitis occurring following grain overload or an overload of lush young grass. Platelet-activating factor (PAF) is a mediator of endotoxaemia, but whether PAF receptor antagonists play an important role in equine laminitis control is uncertain. Endotoxaemia and lactic acidosis are also implicated in obstructive bowel disease and equine colic precipitated by abrupt increases in the intake of starch and protein.

Endotoxaemia

The horse is particularly sensitive to endotoxins and endotoxic shock can be fatal. Endotoxins are lipopolysaccharides (LPSs) which are a structural component of the outer bacterial cell wall of the Gram-negative, nonsporing rods of the Enterobacteriaceae, including *Escherichia coli*, inhabiting the intestines. LPSs are habitually present in the intestinal contents (as much as 80 µg/ml). In fact, repeated administration of sublethal doses of LPSs results in attenuation of the host response. Moreover, both early- and late-phase endotoxin tolerance have been recognized (Allen *et al.* 1996). This tolerance not only provides protection in some individuals, but may also point to ways of providing prophylaxis. However, large doses of LPSs are toxic. Clinical signs of endotoxaemia are mediated by prostaglandins which seem to inhibit gastric-acid secretion. Doherty *et al.* (2003) recently reported that intra-gastric infusion of LPSs caused decreases in gastric acid and $[K^+]$ output and an increase in $[Na^+]$ output, partly through the mediation of prostaglandins.

During grain (soluble carbohydrate) overload there is a rapid increase in numbers of lactic-acid-producing bacteria (species of anaerobic lactobacilli and streptococci) concomitant with a decline in intracaecal pH, which may fall from 7 to 4 within 12–24 hours. Starch-fermenting organisms grow much more rapidly than do those that ferment cellulose, and the starch fermenters proliferate at their expense. Organisms that use lactic acid as a source of energy are not present in sufficient numbers to cope with the surge and their numbers may decline as some are unable to withstand the very low pH values attained. The ciliate protozoa, which are much larger and slower growing than bacteria, normally engulf starch, fermenting it at a relatively slow rate. They then act as a starch reservoir, preventing an excessive rate of bacterial starch fermentation, but they are also killed by the acid environment and so no longer function as buffers. As these normal homeostatic mechanisms are destroyed, acid production proceeds at an accelerating rate. The author's own evidence (Frape *et al.* 1982a) showed that numbers of protozoa increased with

increasing starch intake up to a threshold, beyond which there was a precipitous decline in numbers.

De Fombelle *et al.* (1999a) abruptly introduced barley as half of an otherwise forage diet of adult ponies. The barley was given before the forage in each of two meals daily at the maintenance level. The introduction led to an increase in the molar % of lactate and propionate and to a decrease in that of acetate in colonic contents 30 hours later, without a change in the total VFA content. Fibre utilization was depressed, but no adverse effect on health occurred. After 14 days, the colonic pH had decreased from 6.7 to 6.3, although each pony had its own pattern of ecosystem response. Dawson *et al.* (1999) similarly reported no change in total VFA of the caecum in ponies given up to 50% of dietary DE as barley. Again, lactic-acid production increased with increases in the population of lactic-acid-producing enterococci, but there was also a rapid increase in the population of lactic-acid-utilizing bacteria [these probably included *Veillonella* and *Propionibacter* spp., (the author)], so that lactic-acid concentration was lower than with 0, or 30% barley. Hence, the response to the introduction of starchy food can be without untoward effects in many horses.

Where untoward effects occur, many of the Enterobacteriaceae die. Large amounts of LPSs are released into the gut lumen and the integrity of the colonic mucosa is frequently lost. This is likely to be caused by intestinal ischaemia, perhaps abetted by the lactic acidosis, or by previous parasitism. Concentrate overload has led to concentrations of endotoxin of 1–30 µg/ml in the small intestine, but in the large intestine the concentrations can be up to 160 µg/ml. Consequently there is a considerable transmural movement of LPSs, leading to their presence in portal and systemic blood.

In health, endotoxin is cleared rapidly by the Kupffer's cells of the mononuclear phagocytic system within the liver and so plasma levels are normally <0.1 ng/l. The overwhelming of this system, leading to endotoxaemia (plasma levels of 2.5–82 ng/l with two peak concentrations 32 and 48 hours after carbohydrate overload), is associated with early systemic vasoconstriction, tachycardia, arterial hypoxaemia, hyperpnoea, respiratory alkalosis, pulmonary hypertension and fever, followed by an increase in vascular permeability, haemoconcentration, systemic hypotension, an alteration in mucous membrane colour, a prolongation of the capillary refill time, capillary thrombosis, thrombocytopenia and neutropenia with neutrophil sequestration in blood vessels. The decrease in capillary blood flow causes decreased blood perfusion of vital organs, but there is an increased perfusion of the GI tract. The decreased capillary flow and slow refilling of the capillary bed is associated with cold extremities. Incomplete perfusion of the lungs through capillary shunting causes incomplete oxygenation and lowered oxygen tension of the blood (hypoxaemia). This response, added to the restricted blood flow through other tissues and organs, including restricted extraction of lactate by the kidneys, aggravates the situation and contributes to anaerobic glycolysis and further production of lactic acid. A restricted hepatic blood flow reduces the removal and metabolism of lactic acid and diarrhoea causes dehydration, contributing to haemoconcentration.

Endotoxaemia is said to occur in at least 25% of horses with colic admitted to clinics (Moore 1991), and the effects of endotoxin on the colon are very different from their effects on the small intestine. The decreased perfusion of tissues, with a shift to anaerobic metabolism, causes blood lactic acid concentrations of >700 mg/l, which are associated with death in LPS shock. Experiments in which these toxins have been administered i.v., or intraperitoneally, at rates of 2–30 µg/kg BW, have led to an increase in inflammatory circulating eicosanoids (thromboxane, TXB₂, prostacyclin, 6-keto-prostaglandin F_{1α}, and PGE₂) (Ward *et al.* 1987; King & Gerring 1991). The eicosanoids are derived from arachidonic acid, mobilized during damage to the endothelium of blood vessels by endotoxin (see ‘Polyunsaturated fatty acids’, Chapter 5). Horses with intestinal ischaemia have a plasma endotoxin concentration in the range 30–100 ng/kg BW. Plasma endotoxin of 0.1 µg/kg BW (100 ng/kg) leads to an increase in body temperature of approximately 1°C. The synthesis and action of inflammatory mediators are central to the aetiology of endotoxaemia, and potent cyclo-oxygenase inhibitors (which inhibit conversion of arachidonic acid to eicosanoids) have been an effective therapy, if administered promptly. The feeding of fish oil (probably 500 g/day plus vitamin E) as a prophylactic may help.

Where grain overloading has occurred, and endotoxaemia is likely, early therapy is most rewarding. Treatment should also be directed towards preventing laminitis and may include:

- cyclo-oxygenase inhibitors;
- fluid replacement (this is important in treatment, including glucose, and correction of any bicarbonate deficit, after measuring acid–base status). Oral fluid therapy may be adequate in mild cases, but i.v. administration of physiological solutions will be necessary in horses severely affected;
- colloid solutions (increased vascular permeability and loss of blood colloid is a characteristic of endotoxaemia);
- evacuation of the starch overload from the GI tract. Mineral oil, administered by nasogastric tube, assists by decreasing bacterial fermentation, it may slow the absorption of endotoxin and it facilitates evacuation of colonic contents. Lipoid pneumonia has resulted from administration of mineral oil using a nasogastric tube. Tachypnoea and tachycardia are common presenting characteristics and incorrect placement of the tube must be avoided (Scarratt *et al.* 1998; Bos *et al.* 2002);
- virginiamycin treatment (see ‘Lactic acid production and measurement’, this chapter). This treatment is not permitted under EC rules.

Dietary prevention of grain overload

The underlying processes in grain overload indicate that feeding methods should be imposed to foster dietary health. By slowly increasing the concentrate ration, the bacteria that ferment lactic acid and the protozoa that engulf starch are encouraged to multiply (Table 11.2). These increased numbers of organisms act as a substantial buffer against a decline in pH of the large intestine. The concentrate

Table 11.2 Daily ration to be divided into at least three feeds for horses and ponies with laminitis.

	Mature weight (kg)	Concentrate	Concentrate per day (kg)	Grass hay per day (kg)
<i>Pony</i>	200		—	3.50
	300		—	4.25
<i>Horse</i>	500	Micronized barley	4.5 ¹	<i>Ad libitum</i>
		Oatfeed or molassed chaff	1–2	
		Soya/micronized soya	0.5	
		Molasses (beet or cane)	0.4–0.5 ²	
		Limestone flour	0.05	
		Potassium chloride	0.03	
		Sodium bentonite ³	0.05	
		Stabilized fish oil ³ + vitamin E	0.35	

¹Micronized barley is digested preceacally to a greater extent than micronized oats or other cereals.

²If molassed chaff is given, provide only 0.4kg molasses as well.

³These should be helpful in the prophylaxis of laminitis in horses and ponies prone to the disease. The use of fish oil should be considered experimental.

portion of the ration should never be increased by more than 200g/day for a 550kg horse. This means that 40 days should be allowed when raising the concentrate portion of the diet from 0 to 8kg.

Potter *et al.* (1992a) found that low intake of almost any source of starch leads, primarily, to its digestion in the small intestine, but, with larger meals, starch spills over into the large intestine. In order to prevent digestive dysfunction resulting from starch overload to the small intestine, starch intake in horses, given two or three meals daily, should be limited to approximately 0.4% of body weight per feeding. Where the starch is relatively insoluble, a lower percentage would apply. Processes that gelatinize the starch grain, such as micronization, enhance small intestinal digestion of that starch at moderate and high rates of intake and so those processes reduce the risk of overloading. More recent evidence (see 'Laminitis control', Chapter 2) indicates that the non-structural carbohydrate components of feed should be limited to 0.25% of body weight per meal. These components include both starches and fructans, as Bailey *et al.* (2002) showed that dietary inulin and corn starch lowered caecal pH to a similar extent.

Lactic acid production and measurement

The shock of endotoxaemia results in increased anaerobic glycolysis in muscles. The product of this is L-lactic acid, associated with metabolic acidosis. Reliable measurements of this acid require careful, problem-free sample collection and immediate mixing with cold perchloric acid or sodium fluoride. To measure blood D-lactic acid (see below) the samples should be treated with perchloric acid and analysed using D-lactate dehydrogenase. Alternatively, plasma anion gap [sodium plus potassium – (chloride plus bicarbonate)] measurement is free from these problems and is a slightly better prognostic indicator than is lactic acid measurement. It is a good

measure of the accumulation of acid anions (e.g. lactate, ketoacids, phosphate) generally (Gossett *et al.* 1987), and is suitable for routine application.

Lactic acid is, of course, normally produced in the muscles during anaerobic work, as described in Chapter 9. Why then is the lactic acid produced in the intestine potentially more lethal? First, anaerobic work can be sustained for only a few minutes, after which aerobic conditions lead to the complete metabolism of the lactic acid. In contrast, lactic-acid fermentation may persist for 24–36 hours. Second, at least ten species of *Lactobacillus* and *Streptococcus bovis* produce lactic acid as either a racemic mixture of the D(–) and L(+) forms, or in the D(–) form (a few produce the L(+) form), whereas that produced in the muscles is solely of the L(+) type. This latter type is dehydrogenated by lactic dehydrogenase, with the formation of pyruvic acid. However, lactic dehydrogenase in the muscle is unable to catalyse the dehydrogenation of the laevorotatory D(–) isomer, which therefore accumulates in tissues following absorption and so can exacerbate the effects of endotoxin, as its protracted existence leads to greater tissue damage. Horses need a fully functional liver to catabolize large amounts of lactic acid and those that have suffered liver damage through disease and infestation, and from bad feed management, are less able to cope with rapid increases in the dietary energy allowance. The lactic acid accumulating in the hind-gut can also induce diarrhoea. A granular form of virginiamycin (4 or 8 g/kg feed, with 8 kg feed/day), (Rowe *et al.* 1994; Johnson *et al.* 1998), which retains activity in the hind-gut of horses, has been shown to suppress D(–) lactic acid production. J.B. Rowe (personal communication) prevented acidosis, an elevation of blood D(–) lactic acid concentration and laminitis by oral supplementation with virginiamycin at the rate of 0.48 g/100 kg BW per day. This treatment is not permitted under EC regulations.

Laminitis and other diseases of the hoof

Laminitis may be caused by overwork, concussion of the feet, infections, abortion, high fever, drug-induced complications and the consumption of certain toxins, especially where liver function is abnormal. However, by far the most common cause (compounded by inadequate exercise) is the overconsumption of concentrates or of lush young grass by animals unaccustomed to them. After a grain overload, horses seem to be more likely to survive severe cardiovascular stress than are ponies. Ponies seem to have a greater risk of laminitis than do other equine animals.

The consumption of excessive quantities of starch from cereals, and fructans from pasture, leads to incomplete pre-ileal digestion and absorption of carbohydrate. This undigested material is the substrate for rapid fermentation in the large intestine. High concentrations of VFAs accumulate, with a rapid fall in the luminal pH. As indicated above, under these conditions, lactic acid accumulates, as the acid environment favours those organisms producing lactic acid (principally streptococci and lactobacilli, Gram-positive organisms) and the environment is unfavourable to organisms that use lactic acid (unless starch increase is very gradual). The pH decreases even further with damage to the caecal epithelium and a rise in concentration of bacterial endotoxins.

It is impossible to deduce what the threshold level is for starch-induced laminitis, as there is a wide gulf between the starch doses used to induce laminitis (>1000 g starch/100 kg BW) and those doses (as little as 24 g starch/100 kg BW) that can cause a decrease in caecal pH. Johnson *et al.* (1998) gave horses meals providing approximately 1500 g starch (300 g/100 kg BW) and recorded a decrease in faecal pH from 6.7 to 5.9. A decrease in the pH below approximately 6.4 was associated with abnormal behaviour, including wood chewing and bedding consumption (also see 'Gastric ulcers', this chapter). McLean *et al.* (1998b) showed that the feeding of as little as 24 g starch/100 kg BW per meal could cause a measurably lower caecal pH (6.26 v. 6.48 pH), and lower acetate and higher propionate concentrations. Micronized cereal was shown to be preferable to either extruded or rolled barley, as it achieved greater precaecal starch digestion.

Garner *et al.* (1977) reported that, following carbohydrate overload, the greatest increase in blood lactate normally precipitates circulatory collapse and death with or without symptoms of laminitis. Lesser increases in blood lactate are more frequently associated with laminitis and the lowest increases with neither effect (circulatory collapse or laminitis). The development of laminitis is the most frequent outcome of lactic acidosis. In their study, 70% of the cases developed this condition, whereas only 15% suffered fatal cardiovascular collapse. Garner *et al.* (1977) concluded that the rate of increase in blood lactate, as determined by blood measurements at 8 and 16 hours, gives a fair indication of whether the horse will die or will contract laminitis, and so it provides the basis for appropriate therapy. Other work has shown that where arterial plasma lactate exceeds 8 mmol/l, death is inevitable, whereas survival is probable with maximum values below 3 mmol/l (Coffman 1979c).

A survey of 108 laminitis cases by Slater *et al.* (1995) indicated that GI disease, occurring just prior to its onset, is a very frequent cause of acute laminitis. Of the 35 acute cases, the predisposing health problems were: colic 23%, grain overload 23% and grass founder 8%. Generally, the principal associated causes are:

- (1) fat ponies on lush pasture;
- (2) carbohydrate overload;
- (3) endotoxaemia;
- (4) excessive weight bearing on a sound leg and post-exhaustion myopathy;
- (5) stress of exercise in overweight animals; and
- (6) excessive tube feeding of sick, aphagic horses with a high carbohydrate–protein diet.

Causes (1), (2) and (6) are very likely owing to the production of lactic acid of the D(–) or DL type, in the large intestine, from rapidly fermentable carbohydrate.

The first scientific investigation of laminitis was conducted by Obel in 1948, whose name remains linked to the scale describing the severity of lameness. Obel's four grading scales for laminitis are:

- Obel Grade I: feet lifted incessantly and alternately; no lameness at a walk, but at a trot horse moves with a short stilted gait;

- Obel Grade II: horse is willing to move, but gait is characteristic for laminitis. A forefoot may be lifted without difficulty;
- Obel Grade III: horse moves reluctantly and vigorously resists attempts to lift a forefoot; and
- Obel Grade IV: horse moves only when forced.

Laminitis of Obel Grades I to IV occurs with an unerring frequency following endotoxaemia. Laminitis is the local manifestation of a serious metabolic disturbance. The front feet are frequently the most severely affected, and the animal may adopt a straddled stance with the front feet pushed forwards. Often a strong digital pulse can be detected at the fetlock; sometimes petechial haemorrhages are observed in the buccal cavity and oedema seen along the underline of the belly. Abdominal pain (colic) accompanies ischaemia and a loss of intestinal borborygmi. Chronic inflammation of the hoof initiates a more rapid growth of the hoof wall than would otherwise occur so that the toe of the hoof extends and curls up, heavy rings developing on the wall in response to inflammation of the coronary band. Infections can focus on cracks developing between the hoof wall and the sole. Where the angle between the hoof wall and the surface of the third phalanx (pedal bone), measured on X-ray photographs, is excessive, the prognosis is doubtful. The malformation can be partly overcome by rasping and corrective shoeing and any overweight condition should be rectified.

The primary lesion in laminitis is the opening of arteriovenous anastomoses, causing inadequate perfusion of the dermal laminae. The endotoxin-induced formation of microthrombi and venous thrombosis contribute to this failure in circulation. Platelets, critical for thrombus formation, play an important role in the pathophysiology of ischaemic vascular diseases, and so heparin has been shown to decrease the prevalence of carbohydrate-induced laminitis. Bailey *et al.* (2000) found that the incubation of equine platelets in the presence of lipopolysaccharide (endotoxin) and leukocytes caused the platelets to release 5-hydroxytryptamine (5-HT, serotonin), a potent vasoconstrictor of equine digital blood vessels. The leukocytes provide platelet activating factor (PAF) necessary for this release.

Amines also play a vasoactive role and may contribute to the peripheral circulatory disturbances of laminitis. Bailey *et al.* (2002) determined that a caecal overload of either starch or inulin caused a large increase in the amine content of caecal fluid, a response arrested by virginiamycin. Increased blood flow to the foot, recognized by a bounding digital pulse, thus occurs with a shunting of blood away from the laminae. The ischaemia, and lack of nutrition, of the laminae eventually cause a degeneration of the bond between the hoof and the pedal bone (third phalanx), which sinks and rotates owing to the horse's weight, initiating a chronic phase of the disease. In the acute phase it is therefore essential to correct the loss of blood to the laminae in order that chronic damage is arrested. As in humans, feeding horses large meals episodically leads to a transient postprandial plasma hypovolaemia, associated with a loss of sodium and water from the circulation into the GI tract. This could stimulate a peripheral vasoconstriction to maintain systemic blood pres-

sure, but apparently it does not occur in equine digital arteries (Hoffman *et al.* 2001).

Treatment

In cases of grain overload, mineral oil administered by nasogastric tube may slow the absorption of endotoxin. Evidence of pedal-bone (distal phalanx) displacement and rotation should be sought and the animal should be placed on sand or mud, or have frog supports bandaged to the sole. Marked displacement is an indicator of a likely fatal outcome to laminitis. Shoeing, sole support and trimming were commonly part of the treatment protocol for chronic cases. The rapidly fermentable carbohydrate content of the diet should be reduced abruptly and replaced by good-quality hay and/or feed approved for laminitis control, in order to decrease the rate of hind-gut fermentation (also see Chapter 2, General requirements of analytical method). Feed should not be withdrawn completely, as this may cause hyperlipaemia, especially in ponies. Where the dietary Ca:P ratio is low, the addition of limestone to the diet may have both therapeutic and prophylactic effects.

Severe laminitis accompanying endotoxaemia and intracellular lactic acid accumulation is associated with loss of K from muscle cells into the plasma, decreasing the intra- to extracellular K concentration ratio. This causes membrane depolarization, but urinary K excretion may not necessarily increase (nevertheless, assessment of urinary K:creatinine clearance ratios may be useful). K depletion may lead to vasoconstriction of muscle capillaries, causing local ischaemia and hypoxia, anaerobic glycolysis and metabolic acidosis. This sequence may be a factor in equine exertional rhabdomyolysis syndrome (ERS), discussed in this chapter. The fact that both normal and abnormal plasma concentrations of K have been detected in ERS may simply reflect the difficulty experienced in assessing K status and its *in vivo* intra- to extracellular ratio. The ischaemic damage to muscle-cell membranes results in excessive loss of cellular K. Thus, plasma K may be normal, raised or lowered.

The maintenance of normal cellular K concentrations depends on the integrity of energy-yielding systems. Intracellular K leaks from red blood cells into plasma in the absence of adequate glucose and analysis of whole blood should be conducted within two hours of collection. In acute cases of K loss, i.v. dosing with limited amounts of K is appropriate, but must be carried out slowly while cardiac action is continuously monitored. Abnormal electrocardiographic changes can occur at plasma K concentrations of 6.2 mmol/l and severe cardiotoxic effects have been reported at 8–10.1 mmol/l. Molasses (cane or sugar beet) and potassium chloride are recommended to be given with the hay in cases of K depletion, as the vasodilatory effects of K could be helpful.

Fat ponies and horses with a history of laminitis should be given grass hay and should not be turned out onto lush pasture. Individuals presenting signs of laminitis should be removed from pasture, or concentrates, and the feed then restricted to coarse hay (Table 11.2). The hooves should be radiographed for evidence of pedal bone rotation and the individual placed on soft sand or wet soft earth, or frog

supports may be applied. Severe restriction of the energy intake of ponies may precipitate hyperlipidaemia.

Nitric oxide

Fundamentally, acute laminitis is a vascular disease associated with areas of ischaemia or haemostasis within the hoof. A key to this is a failure of the arginine–nitric oxide (NO) system. NO is produced by the action of NO synthase on its substrate, the amino acid L-arginine. NO relaxes vascular smooth muscle to cause vasodilatation. L-arginine, administered i.v. at the rate of 0.42 g/kg BW, as a 10% saline solution, given, in turn, at the rate of 1 mg/kg BW/min, caused immediate reperfusion of laminal tissue in an acutely laminitic pony (Hinckley *et al.* 1996). The involvement of NO was further implied when glyceryl trinitrate paste applied topically to the pasterns of an acutely laminitic pony reduced the ‘bounding pulse’ in the treated limbs, reduced lameness and lowered systemic blood pressure. It is too early to suggest that a dietary supplement of L-arginine for animals at risk may be a preventive approach. L-arginine is likely to be a semi-dietary essential amino acid in the horse. This means that the horse may not be able to synthesize sufficient from other amino acids. The dietary requirement is probably in the region of 25 mg/kg BW daily, so that daily supplements of the order of 5–10 g for a 500 kg horse might be considered. This is only 3% of the dose given to a laminitic pony and so may have no preventive effect for this and other reasons.

Thyroxine (T_4)

Abnormal blood T_3 and T_4 concentrations have been observed in horses affected with laminitis. The values may be depressed during the two days prior to the onset of lameness and horses with chronic laminitis have elevated serum T_3 levels. These effects, and a reduced insulin sensitivity, are considered to be a consequence of laminitis and not a cause. In cases of equine goitre, associated with depressed plasma T_4 levels, the recommended approach is to replace existing feedstuffs with feeds of known quality and to treat affected animals with thyroxine. The reason for this is that any hypothyroidism may have resulted, among other causes, from excessive, or inadequate, dietary iodine. If either of these is a cause it should be established by dietary analysis before dietary changes are invoked.

White line disease

The hoof capsule consists of the hoof wall, sole, frog, bar and white line. The white line is located between the *stratum medium* of the hoof wall and the sole. It is softer than the hoof wall or the sole, and so is able to help dissipate the stresses of the hoof in motion that act to separate the wall from the sole. Disease of the white line, thought to be contributed to by nutritional problems, is visible as dark, discoloured regions of the white line, but these may remain unnoticed under the horseshoe. If left untreated, hoof-wall separation and infection eventually occur. Kuwano *et al.* (1999) discovered 11.5% of TB racehorses in Japan with the disease. It was more frequent in the fore- than in the hind-hooves and the incidence increased with age.

Some other causes of lameness

Lameness during training is not clearly related to diet, although poor Ca status can increase the risk of stress damage to long bones. Hardness and other features of the surface on which the horse is trained seem to be critical. A good, well-formed turf is protective, probably through a greater compliance compared with dirt. Moyer *et al.* (1991) reported that the incidence of dorsometacarpal disease (buck shins, sore shins, shin splints and stress fractures) was less in horses training on wood fibre than in those training on dirt. The wood fibre was a more compliant surface. There was a decrease of approximately 10% in stress and strain on the metacarpus (force per unit area) during fast work in horses on the wood fibre.

Osteochondrosis

Osteochondrosis (OC) is discussed in Chapter 8. The effect of OC in a joint on susceptibility to lameness depends greatly on the joint affected. Some reports on horses in training indicate that OC of the tarsocrural joint causes a degree of movement disturbance, whereas others have reported no relationship. OC of the stifle joint, on the other hand, is more frequently associated with clinical signs of lameness.

Colic (abdominal pain) and related disorders

Characteristics of colic

Tinker *et al.* (1997a,b) detected an incidence density rate of 10.6 colic cases/100 horse-years and a mortality of 0.7 colic deaths/100 horse-years. The highest incidence was amongst horses of two to ten years of age, with TBs having the highest and Arabians the lowest breed-specific incidence rates. More than one change in hay feeding, changes in concentrate feeding and feeding high levels of concentrates yielded the highest risks. The provision of whole grain reduced this risk, supporting the well-held view that excessive intake of readily available carbohydrates is a major cause.

Many colics involve the presence in the stomach, or intestines, of a thick, sticky mass of fermenting feed or a compacted mass of roughage. Colic may wax and wane in concert with intestinal smooth muscle contractions and the pain is present in several abnormal conditions. As this implies no diagnosis it is apposite to discuss the various types and causes of colic and the management favouring a healthy prognosis. Probably all equine animals experience colic several times in their life, so that in various degrees of severity it is very common and in its most severe forms is associated with disorders which are the most common causes of death. Records show that 80% of cases recover spontaneously within one to two hours, but in the remaining 20%, unless immediate action is taken, a disturbance that may initially be mild can become fatal. Colic usually accompanies a rise in blood lactate, and the severity and outcome are closely correlated with this increased value. Lactate con-

centrations in the peritoneal fluid are also typically higher than in the blood, except for cases of impaction.

Most colics are characterized by some of the following postures and reactions in various forms and intensities: tail twitching; pawing the ground and restlessness in which the horse may get up and down frequently; playing with its food and water; submersing the nostrils and blowing bubbles; and generally losing appetite. The head is frequently turned towards the flanks and, in the extreme, the horse rolls and thrashes about, risking further damage. However, one might enter the box to find the horse cast, with no intestinal sounds, no droppings, or a very few small ones, and a much-distended abdomen. Frequent staling (urination) may be attempted in an endeavour to relieve pressure on the bladder. The rapidity of heart beat and respiration rate and the extent of sweating and fever will depend on the severity of the disorder. Normal heart rate is generally 38–40 beats/min but the rate may rise to 68–92/min in moderate colic and to over 100/min in severe pain. Similarly, respiration rate, normally 12–24/min, may exceed 72/min and the normal body temperature of $37.7 \pm 0.3^{\circ}\text{C}$ ($100 \pm 0.6^{\circ}\text{F}$) will be elevated. Other signs can include diarrhoea with undigested cereals in the faeces, a foul-smelling breath, ingesta in the nostrils, frequent stretching, and, occasionally, skin changes in the form of a nettle rash. Capillary perfusion time is increased, as measured by thumb pressure on the gum, after which the white patch regains its colour over a longer period than the normal 1–2sec. Dehydration is also expressed as a delay in the return of the skin to its normal posture after being pinched.

Clostridia

Excessive consumption of concentrates favours the rapid multiplication of clostridia. Equine intestinal clostridiosis (enterotoxaemia), resulting from the rapid growth of *Clostridium perfringens* type A or D in adults and *C. perfringens* type C in foals, has been described. *C. perfringens*, which secretes an enterotoxin, may be a normal inhabitant of the gut, although recent evidence may indicate otherwise. Several groups (Båverud *et al.* 1997, 1998; Weese *et al.* 2001) have observed that in approximately a quarter of adult horses and foals presenting with intense abdominal pain, or diarrhoea and enterocolitis, *C. perfringens*, *C. difficile*, or their toxins, can be shown to be present. In very few healthy controls were these pathogens present. It is possible that in small amounts *C. perfringens* toxin may be harmless, but, when there are large numbers of this species, excessive gas is produced and the toxin causes damage to the intestinal mucosa and precipitates diarrhoea. The toxin is neutralized by antibody, but immediate therapy involves the replacement of depleted tissue water and electrolytes and the relief of GI tympany (ballooning).

Recently, Båverud *et al.* (2003) confirmed that *C. difficile* was not only associated with acute colitis in mature horses, following antibiotic treatment, but it was present also in many healthy neonatal foals. They also confirmed that it survived for at least four years in equine faeces.

Gastric ulcers

(See also 'Gastric lesions in foals', Chapter 7.) More than half of TBs in training may suffer from gastric ulceration. Lesions lie in the nonglandular *pars proventricularis*, particularly the region adjacent to the *margo plicatus*. Lesions were present in the gastric mucosa of nearly all TB horses in training examined by Murray *et al.* (1996) and those in the glandular mucosa were much less severe than those in the squamous mucosa, which became particularly severe as time in training and racing progressed. In Sweden the prevalence is highest amongst Standardbreds and TBs (Sandin *et al.* 2000).

Signs generally include periprandial colic, bruxism, ructus and reflux. It is considered that gastric-acid hypersecretion, gastric-emptying disorders and disturbances in gastric mucosal blood flow are potentially involved in the initiation of gastric ulceration. The combination of bile salts and gastric acid is more damaging to the squamous mucosa than the acid alone, so that fasting for as little as 12–24 hours presents a risk, increasing, as it does, the gastric bile-salt concentration (Berschneider *et al.* 1999).

Stress-associated catecholamine secretion may result in sufficiently frequent vasoconstriction, hypoxia and inanition of the mucosa to precipitate the lesion.

An alternative explanation relies on the fact that when concentrates enter the stomach the pH remains high for longer than with hay, so that considerable fermentation occurs. However, as the pH falls, acetic, propionic, butyric and valeric acids become undissociated (pK_a values 4.7–5.0) and lipid soluble. This solubility is likely to be greater for the longer carbon chains of butyric and valeric acids. In this state valeric and, to a lesser extent, butyric acids, in particular, diffuse into and acidify the non-glandular mucosal cells, damage Na^+ transport and barrier function, causing cell swelling, necrosis and ulceration (Nadeau *et al.* 2001). Valeric acid even manifested its adverse effects at pH 7.0 (Nadeau *et al.* 2003).

These workers (Nadeau *et al.* 1999) previously found that the gastric pH was higher, and ulcer lesion severity was lower, in mares given a diet of alfalfa hay and grain cf. those given brome grass hay during the initial five hours after feeding, when the gastric acetic acid concentration was highest. The high Ca content of alfalfa may have arrested an initial fall in pH, so that the VFAs remained ionised until they flowed into the duodenum.

After ulceration, gastric acid prevents healing of the mucosa and therapy has successfully included H_2 -receptor antagonists. Work in the USA showed that omeprazole (a proton pump inhibitor) given to TBs at the rate of 4 mg/kg BW per os per day for 30 days followed by 2 mg/kg BW per day for the next 30 days led to a reduction in ulceration of the squamous region of the stomach and improved race performance (Nieto *et al.* 2001; Johnson *et al.* 2001). Trainers indicated that ulceration was associated with poor appetite and performance, a dull coat, loose faeces, loss of weight and disinterest in exercise and that the response to omeprazole was an improvement over that to cimetidine.

Although there is some association of gastric ulceration with the consumption of

large quantities of concentrate feeds, the absence of some environmental stresses among horses involved in light work or pleasure and those on pasture may contribute to a lower prevalence in these situations. Thus, the absence of frequent stress reactions, together with the lower dry matter and pH of gastric contents in horses receiving bulky feeds, could be important preventative factors. De Fombelle *et al.* (2003) found high gastric concentrations of lactobacilli and lactate-utilizing bacteria present following a large cereal meal. Although no association of these bacteria with gastric erosion has been demonstrated, it may be prudent to reduce the rate of feed consumption and to provide several regular, small, daily meals of an open texture, allowing rapid penetration of the feed by gastric acid with its arrest of microbial fermentation. Moreover, intermittent deprivation of feed, given as hay, for periods of 24 hours, causes erosion and ulceration of the squamous epithelium. Gastric ulcers in human subjects have an association with the presence of *Helicobacter pylori*. The higher postprandial gastric pH in concentrate-fed horses may be more conducive to the survival of this, or a related, bacterium, although it tends to be protected from an acid environment by urease secretion. If survival is enhanced by a raised pH then the use of H₂ receptor antagonists could be counter-productive in the longer term.

Ill health associated with physical and microbial quality of feeds

The physical quality and hygiene of all feeds used for horses is important in maintaining good health. If the concentration of moulds, yeasts and bacteria in cereals is high, then there is increased risk of digestive disturbances and of respiratory disease. A high feed-yeast population may be associated with increased risk of gastric colic and tympany. A high feed level of lipopolysaccharides (*Enterobacteriaceae*) is associated with health disturbance, and *Salmonella* in silage can cause fatal colic (see 'Silage and haylage and their safety', Chapter 10, and later this Chapter, for botulism). Chopping hay and straw too short can increase the risk of colic, and excessive stickiness of concentrate feeds (e.g. high gluten content) seems to cause gastric colic. Moulds, including ergot, can cause severe respiratory disease (Meyer *et al.* 1986).

Oesophageal impaction (choke)

Greediness, poor dentition, inadequate water, foreign bodies and the consumption of coarse bedding may predispose the horse to oesophageal impaction: a foreign object, or feed, lodging in the oesophagus. Normally, the obstruction will clear after a while and choke may be common in individuals possessing gullets with an abnormal structure. Long-standing impaction can result in chronic damage to the wall of the oesophagus and spasm, causing recurrence of impaction, dysphagia, coughing and regurgitation of food from the nostrils and mouth. There may be enlargement of the cervical oesophagus. Food withdrawal and immediate veterinary action is vital for successful management. In simple cases, sedation and nasal intubation with

a small warm water lavage and external massage frequently works. Rehydration with isotonic fluids given i.v. and other veterinary treatment follow.

The obstruction is sometimes caused not by physical impaction of the oesophagus, but by an absence of adequate *free* saliva in the throat. Thus, the faster a horse eats, the more likely is choke to occur. The implication is that softer feed pellets and sugar-beet pulp pellets are more likely to be causes owing to the absence of large amounts of free saliva, in comparison to the effects, in horses with good teeth, of a harder pellet that requires more chewing, or whole cereal grain that does not absorb much saliva.

It has been calculated that the intestinal tract of the average horse holds about 100l fluid, that in water deprivation – as may be induced by choke – is drawn on to maintain homeostasis. If choke prevents feeding for more than six to seven days, dehydration precipitates prerenal azotaemia (raised blood urea). Once the obstruction has been removed, the horse should be watered and fed several times per day with small quantities of wetted nuts or other feed, and stones the size of tennis balls should be placed in the feed box to retard the rate of feed consumption. Some scarring of the oesophagus will have occurred, and this may cause repeated trouble. These horses should be given soaked feeds.

Gastric impaction

There is little evidence concerning the causes of gastric impaction, although the ingestion of coarse roughage and inadequate water intake may contribute to it. A stomach tube should be placed to allow the expulsion of gases and to permit the administration of antifermentatives such as chloral hydrate or turpentine (an oil obtained from various species of *Pinus*) in raw linseed oil, although surgery may be needed. Liquid paraffin may be given for impactions by nasogastric tube at rates of 2–6l/500kg horse once or twice per day for several days, and 0.5–1l of raw linseed oil (acting as an emollient cathartic) may be used in obstinate cases together with warm salt water to stimulate thirst. For flatulent colic 30–60ml turpentine may be added to the oil.

Intestinal impaction

The aetiology of ileal impactions is also unknown, although ascarid impactions in foals and tapeworm infestations are less common causes. Large amounts of coarse roughage feeds or excessive cereal intake may contribute to the risk, when ingesta may be present in the nostrils.

Normal gut movement in cases of impactions of the large bowel is encouraged by cold-water enemas and massage of any impaction at the pelvic flexure via the rectum. The general use of tranquillizers and pain-killing drugs removes the need for continued forced exercise to prevent the animal from damaging itself and they reduce the risk of a simple impaction becoming a volvulus (twisting of the intestine on its mesenteric axis). Walking may only be necessary to distract the horse's

attention where drugs are unavailable or ineffectual. Quick action in mild colic can forestall a more serious derangement precipitating endotoxic shock and death.

Spasmodic colic

In spasmodic colic there is an increase in bowel movement, which may be precipitated by a sudden change of feed, work and chilling. Spasms may last for a few minutes or up to a half-hour, and may occur repeatedly over a period of hours, the typical signs being those already discussed (see 'Colic and related disorders', this chapter). Recovery occurs without treatment, but relief of pain and the use of spasmolytic drugs is helpful in amelioration. The colic is associated with increased parasympathetic tone and, from experience, flighty horses are subject to spasms of this kind.

According to Meyer (2001) large intakes of concentrates per meal, little forage and a low standard of hygiene of the feed are major dietary causes. These conditions stimulate excessive microbial activity in the stomach and duodenum, with gas, organic acids and especially lactic acid seeming to play pathogenic roles. The lactic-acid content of the ileal chyme was greatest following the feeding of whole grains as oats, followed by barley grain and least as maize grain, although from other evidence whole grain is preferable to ground cereal.

Parasitic worms

Strongyle larvae cause damage to the lining of blood vessels, particularly that of the anterior mesentric artery and its branches, and this can lead to various degrees of occlusion and inhibition of blood flow (ischaemia). Thromboembolism can be a major contributory cause of the complete loss of blood flow to, and death of, a portion of the intestinal tract, leading to obstructive colic. Where the blockage is incomplete, recurrent colic will be experienced. In acute cases, surgery is needed, but thorough worming at 30–60-day intervals will help to contain the situation. A defined dose of specific wormers (Table 11.1), given under veterinary guidance, will have some beneficial impact on the larval stages. (These stages cause a rise in intestinal alkaline phosphatase activity of peritoneal fluid; see Chapter 12). In young horses, impaction of the small intestine with ascarid worms can occur where management is bad, necessitating immediate anthelmintic treatment.

Ileal impaction (ileus) and colonic impaction

Ileal impactions are defined as intestinal obstruction, with the accumulation of fluid and gas, as a consequence of loss of smooth-muscle activity and associated loss of peristaltic movement of digesta. Hillyer & Mair (1997) and Mair & Hillyer (1997) reviewed features of chronic and recurrent colic. Colonic impaction was a major cause of chronic colic. Verminous arteritis was one of several causes of recurrent colic.

Solid food or water, given orally, in cases of small-intestinal ileus, will aggravate gastric distension and should be withheld. Isotonic i.v. fluid treatment, together with intestinal lubricants, may be indicated. Surgical intervention is usually necessary where obstruction is complete.

About 30% of all colics are in the form of intestinal impactions, and, of these, most impactions occur in the large intestine. Steel & Gibson (2001) reported that the most common gastrointestinal causes of colic in pregnant mares were large colon impaction, displacement and torsion. As briefly mentioned in Chapter 1, impactions typically are located either at points where there is a change in the diameter of the colon or at flexures where it turns acutely. More frequent sites are the pelvic flexure and where the right dorsal colon empties into the small colon, but occasionally the sternal and diaphragmatic flexures may be involved. Impaction may also occur at the ileocaecal valve. The closer to the ileum the large-intestinal blockage occurs, the more dangerous it is, as it will severely restrict water resorption in the caecum and ventral colon, and this can lead to dehydration and hypovolaemic shock.

The horse suffering colonic impaction will frequently look at its flank, emit no intestinal sounds and void small, mucus-covered droppings; palpation of the impaction is frequently possible. Inadequate water intake, excessive sweating and hard exercise together with excessive amounts of coarse roughage are also thought to contribute to colonic impactions. An association with positive *Salmonella* spp. faecal cultures has been observed, which may indicate a reaction to bowel inflammation. Impactions of the caecum seem to be related to several disease conditions including endotoxaemia. Impactions are fairly common in old horses with bad teeth restricted to poor-quality hay with little water after experiencing lush grass. Impaction in the small colon may also occur in foals between two and six months old when roughage feeding is initiated.

Sand colic

Intraluminal obstruction can also result from concretions of hair and of plant material, and combinations of these, and from the horse chewing objects in its environment. These concretions typically occur in the small colon and form as a mineral precipitate on the surface of the material during passage through the colon, over an extended period of time, before the obstruction occurs. The clinical signs include abdominal discomfort, distension and straining to defecate. Treatment for small colon impactions includes the veterinary introduction of gentle warm water enemas with a lubricated nasogastric tube through the rectum.

Sand colic is probably the most common cause of colic in areas of very sandy soils. Sand build-up in the GI tract has been responsible for up to 30% of colic cases in sandy areas of the Southern USA. The mean intake for eight individual mature horses varied from 2.5 g/day to 272 g/day over five days when they were each offered sand in a bucket. However, its elimination from the GI tract is assisted by the consumption of hay (Lieb & Weise 1999; Weise & Lieb 2001). Some horses contract

the bad habit of consuming large quantities of sand and soil. The problem therefore can frequently recur and is associated with periods of inappetence, diarrhoea, anxious pacing up and down with groans on lying down, pawing the ground or a crouched stance with a turned head. Sand may be present in the droppings. Treatment includes repeated large doses of liquid paraffin. Occasionally, however, enteroliths (large stones) are formed, apparently on a nidus of ammonium magnesium phosphate. This gradually enlarges, and its removal requires surgery. Observation may indicate that the horse has a predilection for chewing some material in the surroundings. The solution could then lie in changing the environment and reducing boredom.

Horses with sand impaction of the large colon respond to psyllium hydrophilic mucilloid, given at the rate of 400 g/500 kg BW daily for three weeks, divided among three daily feeds. Flavoured psylliums are available for reluctant patients. Over the period it should remove most of the sand. This treatment may require repeating every 4–12 months. Horses likely to eat sand should be kept on a thick sward and hay should not be offered on the ground, but from racks, in order to reduce the risk of recurrence.

Foal colic

Foal colic is very common in the first two days after birth, and is caused by the meconium blocking the large intestine at various levels. Lubrication of the impacted mass with orally administered liquid paraffin (200 ml) or glycerol, the use of enemas of soap and water and the relief of discomfort are normally sufficient remedies. If no response is registered within a few days, volvulus or intussusception may be suspected. Abdominal pain can also occur at this age through rupturing of the bladder, which is effectively repaired by surgery, and in older foals discomfort may coincide with eruption of permanent teeth. Umbilical hernias can cause colic in young foals, but these usually correct themselves by six to eight months of age. Inguinal hernias, especially in colts, also have similar effects.

Lavoie *et al.* (2000) describe a transmissible enteric disease of foals and other species, caused by *Lawsonia intracellularis*, with faecal transmission. Signs include depression, rapid weight loss, subcutaneous oedema, diarrhoea and colic. The protection given by colostrum seems to be important.

Oral and dietary treatment

Unless one is very familiar with the sequence of events in a particular horse, veterinary help should be sought immediately signs of colic are observed. Feed should be removed *until the cause has been determined*, but clean water should be provided. If the animal shows signs of injuring itself through violent actions the horse may be walked; otherwise leave it alone in a box, free from projections or structures that might endanger it. In all colic cases the horse should be kept warm in cold weather and during recovery a warm bran mash is helpful. The general use of

a mineral oil or a kaolin–pectin paste is safe and will accelerate the expulsion of the offending masses. General treatment also has the objective of preventing rupture of some part of the GI tract, or displacement of its parts by control of pain and tympany, by evacuation of the bowels, by arresting rapid bacterial fermentation and by re-establishment of normal peristalsis. An enema of 9–13 l (2–3 gal) warm soapy water is frequently given in addition to the lubricant mineral oil for intestinal impactions. A mild soapy-water enema is particularly helpful in cases of colonic impactions in foals. Where there is impaction of the large colon, or caecum, oral fluids, including solutions of electrolytes and glucose, may be allowed, but solid food should not be given until the impaction is passed.

From the dietary point of view, therapy includes withholding normal feed, but allowing access to water if there is no nasogastric reflux. Rehydration of the horse both per os (in the absence of reflux) and i.v. is essential, and laxatives are normally given by nasogastric tube. With severe intestinal impactions, a 500 kg horse should be given 6 l fluid every two hours through an indwelling nasogastric tube. Mineral oil can be administered to facilitate passage after the impaction begins to resolve. Following resolution of the impaction, and in the absence of perforations, feeding should be resumed gradually to avoid an immediate recurrence. As soon as GI function resumes, bran mashes are preferable for the first 24–48 hours. Following this, leafy hay may be introduced gradually, along with concentrate mashes, or short grazing spells on a halter should be offered. With prolonged dysfunction, partial or total parenteral nutrition may be required.

Forage chopped very short is said to increase the risk of ileal obstruction. Grinding of forages reduces digestibility, as it tends to accelerate rate of passage through the GI tract. The main advantages of pelleting may be a reduction in dustiness, increased keeping quality of ground material and a reduction in bulk for storage.

Gas or flatulent colic and its treatment

Gas or flatulent colic may be secondary to an obstruction or an impaction and is extremely painful. Distension of the small bowel is rarely noticeable and if the abdomen is unusually large, tympany of the ventral colon may well be present. Sometimes, on post-mortem examination, several regions of the GI tract appear to be involved. An impaction and lack of movement of the intestines inhibit expulsion and minimize absorption of gas into the blood. The latter route of removal is more important than may be realized, as about 150 l carbon dioxide and methane can be absorbed daily from the intestinal tract.

If gastric tympany occurs, the condition becomes evident within four to six hours of eating. Intubation with a nasogastric tube to relieve the pressure is essential, to prevent rupture of the stomach. A warm 4% salt solution, which decreases the viscosity of the fermenting mass and encourages water consumption, is sometimes administered in small quantities at intervals by allowing it to drain into the stomach through the tube. The horse may have adopted a typical dog-sitting attitude, or it may stand without moving the feet, especially where a rupture or a serious intestinal

infarct exists. After stomach rupture, in particular, ingesta is sometimes observed in the nostrils. With different intensities of gas colic the horse may feel cold, despite experiencing a fever, it may exhibit congested mucous membranes of the eyes and have a sour and vinegary breath. Powerful analgesics prevent violent rolling and self-inflicted trauma. Where there is risk of violent action the horse should, if possible, be kept on its feet, but quiet horses are best left alone.

Gas colic is frequently the sequel to a rich diet of cereals, or lush legumes, or even to the inadvertent consumption of a pile of grass cuttings. Quick action is essential, as again violent reactions on the part of the horse may lead to ruptures or to twisting of the gut, with a poor prognosis.

Colic associated with torsion, twists and rotations

Torsion (rotation on its own axis), volvulus (twisting on the mesenteric axis) and intussusceptions (infolding), usually of the terminal ileum into the caecum, all require immediate surgery, and the prognosis must be considered to be poor. The membranes of the eyes and lips are typically dry and pain induces a rapid rise in pulse and respiratory rates so that the loss of carbon dioxide causes alkalosis despite a raised blood lactate. The value and risks associated with post-operative parenteral nutrition have been assessed recently by Durham *et al.* (2004).

Other colics

Abdominal pain is not the reserve of the intestinal tract and may result from bladder and kidney stones, urinary infections, pericardial effusions and sometimes liver disease.

Predisposing factors in colic

- ‘overheating’ – sudden access to large quantities of cereals or stands of green clover and lush grass;
- stress caused by changes in routine, changing stables, and mares and foals in new surroundings;
- irregular work, horses standing idle on full feed or changes in the timing of feeds;
- working a horse on a full stomach. Even during protracted slow work large feeds should not be given and bulk feeds should be excluded. Bulky feeds should be given in the evening, waiting until the digestive powers have been restored by rest;
- work itself can precipitate colic, especially towards the end of an exhausting day – both feed and work should be regular. Extended work should be interrupted by short rests every two to three hours when a few mouthfuls of concentrated feed and water are provided;
- the consumption of excessive amounts of cold water after severe and hot work before the horse has cooled down and/or providing heavy feed at this time;

- insufficient good-quality roughage or mouldy corn and mouldy silage;
- large quantities of cut green feed;
- unfit horses changed abruptly to an increased work rate and a concentrate-rich diet;
- failure to provide fresh clean water at all times;
- lack of teeth care. ‘Quidding’ may be noticed in which small balls of partly chewed feed are dropped into the manger. This is usually associated with cheek teeth that require rasping;
- greedy feeders that bolt feed, or greedy bullies in group-fed herds; and
- inadequate work control.

Prevention

- Each horse has its idiosyncrasies so that before a new horse is placed gradually onto a rich working diet its habits and particular requirements should be studied and understood.
- Hard-worked animals should receive a small feed of concentrates at frequent and regular intervals, and the time of meals should be constant, even at weekends.
- Increased demands for energy should be met by an increased feeding rate of concentrates of no more than 200 g/day in a 500 kg horse and a proportionately lesser rate of increase in smaller horses.
- A sensible and regular exercise programme should be instituted.
- Where a horse is changed from one stable to another, its old routine should go with it and, if necessary, be changed gradually.
- All animals should be checked last thing at night.
- Only good-quality roughage, free from contaminating weeds, and, in the USA, also free from blister beetles, should be provided.
- Stores of horse feeds and dangerous chemicals should always be held in rooms the doors of which cannot be opened by horses.
- No horse should be overworked and, following strenuous work, no substantial feed or water should be given until the animal is cool and rested and then should only be given in moderate amounts.
- Cribbers or wind suckers (swallowing of air into the stomach) (Plate 11.1) should be fitted with cribbing straps, or, if cribbing is known to precipitate colic in the individual, surgery may be necessary.
- Teeth should be inspected at regular intervals. If may be necessary to file the upper and lower molars and premolars or remove teeth with infected roots – decaying teeth may be inferred from excessive salivation.
- Individuals that bolt their concentrate ration should have it mixed with chaff or dry bran. The placement of stones the size of tennis balls in the manger may retard the rate of feed consumption.
- A proper worming programme is essential in all horses and a pasture rotation system should be instituted where horses have access to grass.



Plate 11.1 A 'wind-sucker' mare (cribber) – a vice in which the incisor teeth grip a solid object, the horse pulls down and swallows gulps of air. Sometimes a leather strap is fastened snugly around the neck just behind the jaw to deter the horse from the practice.

Overfeeding

In addition to the consequences discussed in this chapter, overfeeding can have a number of other deleterious effects in horses. Some of the more obvious are listed below:

- Obesity. This is said to reduce fertility and to present difficulties at foaling in mares, to affect the work rate of horses and to accelerate the onset of fatigue;
- Obese horses that suddenly experience food deprivation, as in a drought, may be subject to anorexia secondary to colic, which sometimes causes hyperlipidaemia (high concentration of blood lipids). Pony mares in late pregnancy or in peak lactation and subject to pasture changes are prone to this problem. Treatment can be problematic and certainly requires veterinary advice. Anorexia may also be a sequel to acidosis;
- The overfeeding of young horses, (a) in particular of colts, (b) when the food is given in separate and discrete meals, and (c) where the ration is unbalanced in respect of its mineral content, may cause bone disorders. The principal one is epiphysitis – typically of the distal epiphyses of the radius, metacarpus, tibia and metatarsus – and recognized by bony enlargements and lipping of the physes;
- Contracted tendons, as previously discussed in Chapter 7, may be associated with overnutrition of energy-rich feeds in late pregnancy (producing too much milk), or overfeeding the foal and yearling; and
- Enterotoxaemia in young horses fed in groups is occasionally precipitated in the largest and most aggressive individual. A flatulent colic occurs in which the

intestines are loaded with rich feed and gas. Signs of dyspnoea and subcutaneous oedema may be presented and the cause is apparently a rapid proliferation of the bacterium *Clostridium perfringens*.

Hyperlipaemia

Hyperlipaemia is a clinical condition of which the subclinical form is known as hyperlipidaemia. In the horse, most of the circulating fat is in the form of very low density lipoprotein (VLDL, of hepatic origin), especially in the post-absorptive state of hyperlipaemia. The clinical disorder is characterized by depression, anorexia, elevated plasma TAG concentrations, lipid infiltration of the liver and hepatic failure. In large horses azotaemia also occurs. Various physiological stresses, fasting, obesity, pregnancy, lactation and depressed feed intake predispose horses to the disease. Gupta *et al.* (1999) kept donkeys and mules off dry feed for ten days. Serum became cloudy by the seventh day, although the donkeys were more adaptable than the mules and recovery occurred by the sixth day of re-feeding. The difference in susceptibility to the condition between the donkey and the pony on the one hand, and the horse on the other, may be because of a greater frequency of insulin resistance amongst ponies and donkeys. Insulin is required for the activation of lipoprotein lipase, which is required for TAG clearance from the blood into adipocytes. Starvation depletes body stores of the minerals P, K and Mg, and alfalfa is the basis of a good diet for recovery. Whilst the addition of corn oil to alfalfa dampens the insulin response, the addition of excessive oil can jeopardise Mg status and recovery of Mg reserves (Stull *et al.* 2001).

Tube feeding with readily digestible carbohydrate in relatively small amounts, initially, can be begun if the animal will not eat voluntarily. Insulin therapy (monitoring blood sugar) is frequently used to promote carbohydrate metabolism and fat clearance and to reduce the activity of intracellular lipase, which mobilizes stored fat to form VLDL (Table 11.3).

The disorder can lead to organ failure and mortality rates of 65–80%. The aetiology is apparently different from the condition in man, where the function of the enzyme lipoprotein lipase is impaired. In the horse, overproduction, rather than

Table 11.3 Glucose and insulin therapy for hyperlipaemia.

	Day 1	Day 2	Day 3	Day 4	Day 5
Insulin (iu/kg) intramuscularly twice daily	0.15	0.075	0.15	0.075	0.15
Glucose (g) per os twice daily	100		100		100
Glucose (g) per os once daily		100		100	
Heparin (iu/kg) intramuscularly twice daily ¹	40–150	40–150	40–150	40–150	40–150
Sodium bicarbonate and fluid ² i.v.					

¹ Blood clotting ability should be ensured and maintained, especially if larger doses are used. Pharmaceutical doses of heparin release lipoprotein lipase into the blood.

² Blood acid–base status should be checked first for a base deficit. PCV and blood urea should be measured and elevated values rectified with Ringer's solution.

defective catabolism, of VLDL is the cause of hyperlipaemia. Plasma VLDLs are elevated in hypothyroidism (N. Frank 2003, personal communication). Treatment should therefore be directed towards use of lipid-lowering agents that reduce VLDL synthesis. Hyperlipaemia in the pony is accompanied by an elevation in plasma FFAs, without ketosis, which may provide the stimulus for TAG synthesis and the secretion of the VLDLs. Thus, there may be scope for the prevention and/or treatment of ponies by lowering this hepatic VLDL production with agents such as niacin, as this reduces adipose tissue lipolysis and FFA flux (Watson *et al.* 1992).

Botulism (forage poisoning in adult horses and shaker foal syndrome in foals)

[See 'Silage and haylage and their safety' and 'Equine dysautonomia (EGS, grass sickness)', Chapter 10.] Characterized by dysphagia, weakness and progressive flaccid paralysis, botulism is caused by the exotoxin of *C. botulinum* that interferes with the release of acetylcholine at the neuromuscular junction. Wound botulism has rarely occurred in horses. In adult horses the disease is caused by the consumption of the toxin, but in young foals the toxin can be elaborated by organisms present in the GI tract.

Treatment of infected foals and adults is difficult and requires intensive care with the use of a specific antitoxin. This treatment will not reverse the effects of toxin already bound at the presynaptic membrane and it should be introduced before recumbency occurs. Tube feeding will be necessary, together with measures to prevent aspiration pneumonia, constipation, corneal ulcers and gastric ulceration. Mineral oil may be added to feed if constipation occurs. Foals are optimally protected by vaccinating the pregnant mare, with the annual booster given one month before foaling. This will protect the foal until two to three months of age. The foal should then receive the three-dose toxoid series starting at two months of age.

Idiopathic colitis, malabsorption and chronic diarrhoea

Severe colitis has been produced in ponies by oral treatment with the antibiotics clindamycin and lincomycin, followed by the intestinal contents of horses dying from naturally occurring idiopathic colitis (Prescott *et al.* 1988). Treatment of three further ponies with lincomycin on its own at the rate of 25 mg/kg BW twice daily for three to five days caused death. A clostridium closely resembling *Clostridium cadaveris* was isolated from the colon of each of these ponies and from one of six horses dying from idiopathic colitis, but not from horses with non-fatal diarrhoea. These data may indicate the importance of maintaining a large mixed culture of symbiont bacteria in the large intestine by mixed feeding, which prevents potential pathogens achieving rapid unrestricted growth.

Chronic diarrhoea is normally of large-intestinal origin resulting from some upset to the normal balance of the intestinal flora. It can follow stress, such as the

prophylactic use of oxytetracycline or some other antibiotics. Where the diarrhoea is of small-intestinal origin, it may be connected with a want of certain digestive enzymes detected by xylose and other tolerance tests (Chapter 12). It should also be remembered that the adult horse loses the ability to digest lactose adequately when about three years old. Large intakes of milk sugar after this time may induce diarrhoea. Chronic diarrhoea can also be prompted by parasitism, mesenteric abscesses or some disorder of vital organs.

The loss of integrity of the gut mucosa associated with protein-losing gastroenteropathy commonly causes chronic diarrhoea. The loss will reduce the efficiency of net absorption of energy and protein sources as well as those of minerals, trace elements and vitamins. The diet should be of the highest quality and rich in these essential nutrients, including leafy hay, to compensate for a reduced efficiency of use. Water and electrolyte losses in young stock suffering from diarrhoea can lead to a rapid decline in vigour unless lost fluid is continually replenished.

Salmonellosis, colitis and many other causes of diarrhoea are associated with rapid transit of digesta through the large intestine. This means that fibre digestion is impaired and that the efficiency of reabsorption of water and Na and K ions is depressed (see Chapter 1). Hyponatraemia, hypochloraemia and hypokalaemia may occur. Blood monitoring and a determination of blood acid–base balance are advised, so that the appropriate electrolyte drinks, and fresh water separately, can be given. In the absence of this and of inclement weather, it may be feasible to turn the horse out to sheltered pasture, as green herbage is a good source of electrolytes. Remember that bacterial diarrhoea may cause contamination of the pasture for a period.

Acute diarrhoea

(See also Chapter 7.) Diarrhoea in the adult horse normally indicates colonic dysfunction and it is typically absent from those with lesions limited to the small intestine (Chandler *et al.* 2000). Severe diarrhoea is frequently caused by *Salmonella* infection precipitated by stresses of transport and particularly by strongyle worm infection. Antibiotic treatment to eliminate *Salmonella* is of questionable value and in fact oxytetracycline may trigger the onset of the infection. Salmonellosis may occur in closely stocked groups after mild winters and is associated with heavy worm burdens. It may be transmitted to foals by adults that are asymptomatic carriers. Suspected cases should be isolated in a box with a very strict programme to ensure that contaminated faeces are not transmitted to other stock. The bacteria are not continuously excreted in the faeces, and careful veterinary examination may be necessary to detect any carriers that are shedding the organisms. Feed and rodents may also be suspect reservoirs of potential infection.

All cases of acute diarrhoea are associated with a critical loss of fluid, K, Na and Cl, and unless replacement therapy is quickly instituted the consequences are rapidly fatal in young stock. Appropriate fluids are listed in Table 9.3 (p. 328). Fluid losses of 20–50l, Na deficits of 2000–6000mmol, K deficits of 700–3000mmol and

bicarbonate deficits of 1000–2000mmol may exist and should be made good in adults and young. Foals may experience absolute losses amounting to 15–20% of that of the adult. In acute diarrhoea where there is hypotonicity and dehydration, the use of hypertonic solutions yields an immediate response. Where the animal is dehydrated and hypertonic, then hypotonic solutions are given. The normal plasma values for Na, K, Cl and bicarbonate in horses are, respectively, 139, 3.6, 99 and 26mmol/l.

Foal-heat diarrhoea

Foal-heat diarrhoea occurs typically at the time the dam's first postpartum oestrus is expected, but no correlation has been established between it and chemical composition, bacterial count or oestrogenic activity of mares' milk (Urquhart 1981). The diarrhoea is normally self-limiting in three or four days, and is probably a secretory diarrhoea. Hypersecretion in the small-intestinal mucosa may overwhelm an immature colon unable to compensate by increased fluid and electrolyte absorption. If a prolonged diarrhoea occurs, fluid and electrolytes should be replaced.

Dehydration and potassium status

Although the carcass of a 500kg horse may contain by weight only 15% as much Na or K as of Ca, on average the 1100–1200g K are subject to a much greater flux than is the Ca, owing to its higher solubility in tissue fluids. The volume and water content of muscle cells and of red cells are modulated primarily by the control of their Na⁺ and K⁺ contents. The cell membrane is relatively impermeable to small cations, that is they diffuse slowly, whereas small anions diffuse freely, but haemoglobin, acting as a large anion, remains as an intracellular entity. However, the equilibrium distribution of charged particles between red cells and plasma differs from that predicted by normal diffusion processes. The slow passive movements of Na⁺ and K⁺ are balanced by an active outward movement of Na⁺ and an inward transport of K⁺ in each cell, mediated by several hundred discrete pumps fuelled by ATP. If glycolytic mechanisms yielding ATP break down, or if the cell membrane is damaged such that diffusion leakage increases, then there is a decline in resting membrane potential and the pump mechanism is incapable of maintaining a physiological cellular environment. That an intact glycolytic pathway in red cells is necessary for the maintenance of a physiological cation distribution between cells and plasma has been amply confirmed in numerous experiments.

It is clear that the horse must maintain cellular K within strict concentration limits to maintain normal health. The measurement of plasma or serum K⁺ concentrations, as a guide to body K status, although frequently done, is misleading. Measurements of large numbers of horses have failed to detect any correlation between serum and cellular concentrations of K⁺ (Frape 1984b; Muylle *et al.* 1984b) and serum contains

on average only 3.7–4.3 mmol K⁺/l, that is, 3.8–4.4% of the mean concentration in red cells. In fact, the extracellular fluid of the body in total contains only 1.3–1.4% of the total body K. In maximal anaerobic exercise, serum K⁺ tends to increase, whereas it has a tendency to decrease in endurance work without comparable changes in the cells.

In severe diarrhoea, the bodily loss of K by a 500 kg horse may approach 4500 mmol (175 g), associated with a fall in red cell K⁺ from 97.5 to 75 mmol/l, but without significant change in plasma K⁺ (Muyllé *et al.* 1984a). Protracted work in hot weather apparently leads in a horse of this size to losses of K⁺ and Na⁺ of 1500–1800 and 4000–5000 mmol, respectively. Thus, the measurement of red-cell K⁺, the concentration of which appears to be well correlated with that of muscle-cell K⁺ (Carlson 1983b), is recognized as a more reliable means of assessing K⁺ status and possibly of understanding the underlying processes obtaining (existing) in set-fast ('tying up') and azoturia.

A fall in red-cell K⁺ concentration below 81 mmol/l is associated with weakness of skeletal and smooth muscle, tremor, and, in severe depletion, with recumbency, cyanosis and eventually respiratory and heart failure. The K⁺ ions released from muscle cells during hard exercise act as potent arteriolar vasodilators and they stimulate cardiorespiratory reflex activity. Thus, there is a close correlation between the extracellular increase in K⁺ and the increase in both muscle blood flow and oxygen consumption and therefore in performance.

The blood flow is insufficient in K⁺ depletion precipitating hypoxia, anaerobic glycolysis and metabolic acidosis. This turn of events is pathological, and the ensuing damage to muscle-cell membranes leads to further cellular K⁺ loss, which cannot be restored by a Na⁺/K⁺ pump deficient in readily available energy. Investigations in Belgium (Muyllé *et al.* 1984b) have revealed an anomalous situation in which about 10% of 436 horses examined possessed a normal red-cell K⁺ concentration distributed independently of the remainder. Their mean red-blood cell K⁺ concentration was 83.8 mmol/l, some 13.7 mmol less than the normal for the other 90% despite similarities in management and diet. Other studies by the same group (Muyllé *et al.* 1983) indicated that, in a smaller sample of 43 horses, 11 were in this low red-cell K⁺ range, and of these 9 were performing unsatisfactorily on the racetrack and had a more nervous temperament. An intriguing observation was made by Hess *et al.* (2003), who provided a concentrate low in K, cf. a K-rich mix, for horses subsequently participating in an endurance ride. This caused them to be less dehydrated, with higher plasma Na⁺, during the ride, so maintaining plasma volume and blood pressure. Thus, the ideal composition of electrolyte mixtures for endurance rides needs further examination.

A dietary K deficiency is unlikely to be more than an occasional cause of K⁺ depletion, although several investigations implicate dietary Mg deficiency as a cause. A 500 kg horse given a diet composed of a 50:50 mixture of grain and hay, may absorb daily 90–100 g K⁺, well above the level required for maintenance. Excess dietary K is rapidly excreted so that a generous dietary content is of no avail in the

acute K^+ depletion of diarrhoea, or of abnormally high sweat loss, when in any event appetite is depressed. Dosing with an appropriate solution is the only reasonable approach in overcoming the worst of the deficit.

K in excess is a moderately potent toxin to heart muscle so that only limited quantities may be given i.v., and cardiac action must be monitored. Intravenous infusion rates of 11.5–13.7 mmol KCl/min bring about plasma K^+ concentrations exceeding 8 mmol/l and consequential cardiac arrhythmias and abnormal electrocardiograms, through a transient but excessive alteration to the gradient of the transmembrane K^+ (Epstein 1984). Thus, the bulk of such dosage must be given orally or by nasogastric intubation. By this route Muylle *et al.* (1984a) administered a solution containing glucose (50 g/l), a commercial amino-acid mixture (0.05 l/l), KCl (5 mmol/l), $CaCl_2$ (3 mmol/l) and made isotonic with NaCl, which was partially replaced by Na acetate according to the acid:base balance of the horse. The quantities given daily were proportional to the deficit calculated from red-cell K^+ values (see Chapter 12 for assessment of red-cell K).

Malabsorption of fat-soluble vitamins

Occasionally the efficiency of absorption of vitamins A, D, E and K is reduced and the most frequent cause may be an interruption to biliary flow by obstruction of the bile duct. The immediate effect is a failure in the blood-clotting mechanism, but this can be overcome by injections of vitamin K. Vitamin K administration is also successful in counteracting the bleeding syndrome of dicoumarol poisoning. Plant sources of this poison are referred to in Chapter 10.

Urticaria

(See also Chapters 5 and 12.) Feed proteins, or peptones, are absorbed from the gut in sufficient quantities to stimulate an immune response with circulating antibodies. However, such antibodies occur in the blood of most horses without clinical signs. Feed allergies are expressed as respiratory and/or skin disorders.

Mycotoxins

Depending on the species of the mould, mycotoxicosis causes digestive disturbances, liver and kidney damage, nervous symptoms, infertility and abortions. The aetiology of several mycotoxin infections has been discussed in Chapters 5 and 10.

Grass sickness (equine dysautonomia)

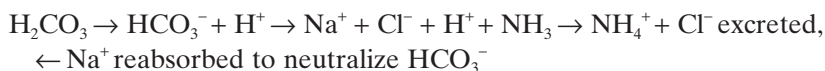
Signs similar to those of choke may be present in grass sickness, but stomach distension is frequently involved, as failure of the normal function of the stomach and of the oesophagus results from neural impairment of the muscles controlling the contraction of these organs. Nasal return of ingesta indicates such oesophageal

impairment. Neurotoxins are apparently a cause (see Chapter 10 for current evidence).

Ammonia toxicity

(See also Chapter 2 for treatment.) Ammonia toxicity is much more likely to occur in the ruminant than in the horse, when the source of ammonia is urea taken orally. The reason for this is that the breakdown of urea to ammonia requires the intervention of urease, which is not found in the tissues of mammals, but is secreted by protein-degrading bacteria in the gut. In the horse, any dietary urea would be absorbed into the blood before it was significantly degraded by these bacteria. With grossly excessive protein (or urea) intake, or when intestinal haemorrhage occurs, protein (or urea) may be bacterially deaminated to the extent that the portal system and the liver are overloaded, and ammonia spills over into the systemic circulation. Otherwise, the liver will 'mop up' ammonia in urea formation, or in transaminations.

Where there is hepatic dysfunction, as in pyrrolizidine poisoning and portocaval shunting of blood, the ammonia will enter the systemic circulation. Its disposal through the kidneys may be arrested by renal pathology. The kidneys would otherwise tend to combat metabolic acidosis by secretion of H^+ ions and ammonia into the tubules, where they combine to form ammonium ions, with reabsorption of Na ions:



Ammonia toxicity has also been recognized in rare cases of unknown aetiology, where equine liver function is normal.

The signs of ammonia toxicity include head pressing, blindness, usually abdominal pain and varying degrees of maniacal behaviour, ataxia and depression. Encephalopathy is the major cause of the behavioural disturbance, which can return to normal following early treatment. Appropriately collected and quickly tested blood samples reveal metabolic acidosis, low plasma bicarbonate (10–15 mmol/l) and hyperammonaemia (150–400 μ mol/l). Where liver function is normal, hyperglycaemia (15–24 mmol/l) and haemoconcentration have been observed, otherwise, with liver dysfunction (detected by raised liver enzymes, bile acids and bilirubin and depressed blood albumin) hypoglycaemia is likely. Blood urea may, or may not, be elevated and blood albumin may be depressed when there has been protein-losing enteropathy without liver dysfunction.

When the source of the ammonia is the intestine, bacterial action is partly arrested with nasogastric neomycin treatment, and polyionic fluids are given to counteract dehydration. Sodium bicarbonate should be reserved for the most severely acidotic cases and given by slow i.v. infusion, as rapid correction of acidosis may increase the intracellular movement of ammonia. K^+ treatment counteracts the

Table 11.4 Mean plasma protein concentrations (g/l).

	Birth	3 Weeks	Yearlings	2 Years	3+ Years	Mares at stud
<i>TB</i>						
Albumin	25	25	27–28	32	34	27
Total globulins	20	22	31–35	25–28	25–35	31
<i>Non-TB</i>						
Albumin			29	27	28	34–37
Total globulins			38–44	44	47–49	37–40

toxicity of ammonia on neuronal cell membranes, and is given as i.v. potassium chloride (10mEq/h) while monitoring heart rate.

Protein-losing gastroenteropathy

The condition of protein-losing gastroenteropathy is characterized by weight and muscle tissue loss, lethargy and diarrhoea, caused by leakage of plasma proteins into the lumen of the GI tract. There is a loss of integrity of the mucosa of the GI tract through gastric, or colonic, ulceration, GI parasitism and enteritis caused by various bacterial infections. It is crucial that the cause of the protein-losing condition is determined and that the appropriate veterinary therapy is instituted. Of the plasma proteins, virtually all the albumin and fibrinogen and 60–80% of the globulins are synthesized in the liver (the remaining γ -globulins are mainly formed in the plasma cells of lymph tissue). Globulin synthesis is faster than that of albumin, for which the half-life is longer, i.e. half-life ($t_{1/2}$) 19–21 days. Thus, in chronic cases, especially with hepatic malfunction, the albumin:globulin ratio and the colloid osmotic pressure of plasma both decline, owing to a failure of liver protein synthesis to keep pace with the loss (normal values are given in Table 11.4).

The rate of plasma protein synthesis can be very high, but it depends, apparently, on the level of amino acids, and, critically, of dietary essential amino acids, in the blood. However, normal post-absorptive plasma free amino acid levels, even with adequate intakes, are extremely variable: lysine is about 15–100, threonine 100–250 and methionine 40–60 $\mu\text{mol/l}$. The ratio of tissue proteins to plasma proteins remains relatively constant at about 33:1. Amino acids from muscle tissues and from the diet are used by the liver in albumin etc. synthesis. Thus, losses are buffered, but the net loss will depend to a large extent on the provision of good-quality dietary protein. Diets containing 140g protein derived from soya and legume leaf are recommended.

Parenteral nutrition

Where detailed advice is required on this subject, readers are referred to Lopes & White (2002), who recorded a critical evaluation of parenteral nutrition that was

provided in 79 cases of gastrointestinal disease. Further references will be found in that report. It was noted that the most common complication of parenteral nutrition is hyperglycaemia and that where possible some oral or nasogastric feeding should continue to encourage normal GI tract function.

Protein-losing nephropathy

In severe kidney disease there can be a large loss of plasma protein in the urine. Dietary protein should be of high quality. Until the kidney disease has been resolved by veterinary treatment there has to be a quantitative balance between the protein intake necessary to stem the renal loss and the amount that would tax the kidneys in urea disposal, causing azotaemia. Dietary protein with an ideal balance of amino acids will minimize urea production.

Urinary calculi (uroliths)

Urolithiasis, often with cystitis, is one of the most common urinary disorders, most clinically apparent in adult males, and the bladder is a frequently affected site (De Jaeger *et al.* 2000). Cystic calculi may be 1.5–20 cm in diameter and up to 6 kg in weight. The crystal growth is driven by supersaturation of the urine, and desquamated epithelial cells or mucous collections serve as a nidus for urolith formation. Chemical composition of uroliths varies somewhat with diet.

A high content of oxalates in pasture is a major contributor, and a rise in urinary pH induces precipitation of Ca phosphates, carbonates and occasionally of Ca sulphates, particularly where water intake is restricted. When high urinary pH is an immediate cause, the dissolution of calculi is facilitated by decreasing the pH with acid salts such as ammonium chloride (NH_4Cl), or with ascorbic acid. Daily doses of NH_4Cl may be 45–100 g. A dose of 0.33 g/kg BW has been shown to lower urinary pH from 8 to 6.2. A dose of 500 g ascorbic acid on each of two days, or 1 kg once, both by gastric intubation, effectively lowered the pH to 4.7.

By contrast, oxalate removal requires an alkaline pH. Potassium citrate (0.1 g/kg BW p.o. daily) will alkalinize urine and may help with calcium oxalate stones, although surgical removal is often necessary. Subsequently, clean water consumption should be encouraged by administering 60 g sodium chloride daily in the diet for at least three weeks.

Intestinal stones

(See also ‘Sand colic’, this chapter.) Excessive dietary Mg and P may encourage stone formation in the gut, and feeding 200–300 g NaCl daily assists in preventing the precipitation of Mg and PO_4 around the nidus of a calculus by stimulating fluid intake.

LIVER DISEASE

(See also 'Seneciosis', Chapter 10.) The liver is central to the intermediary metabolism of both nutrients and non-nutrients, and it is the main line of defence in the detoxification of ingested substances. During browsing and grazing the horse ingests many chemicals requiring detoxification, some of which may damage the liver, leading to clinical liver disease. A frequent example is hepatic cirrhosis, resulting from the ingestion of pyrrolizidine alkaloids (seneciosis) present in *Senecio jacobaea* (see Chapter 10). The risk of damage is increased by several antecedent causes of injury. These include:

- hepatic necrosis caused by *Strongylus equinus* and *S. edentatus* larvae and mouldy hay;
- aflatoxicosis and hepatic lipidosis (fatty change of liver, hyperlipaemia and amyloidosis);
- hepatic metastases from carcinoma of kidney, stomach and pancreas;
- GI disease (obstructive conditions, strongylosis, malabsorption and grass sickness; West 1996); and
- biliary lithiasis.

Liver disease (Table 11.5) causes an inability to metabolize and excrete photodynamic substances in the bile. Reactions to ultraviolet light then occur in unpigmented skin and skin not covered by hair, e.g. the muzzle, eyelids and ears, especially in horses grazing green pasture [see also 'Dried lucerne (dehydrated alfalfa)', Chapter 5]. Liver disease occurs typically in pregnant mares of 5–14 years of age and Welsh and Shetland ponies are particularly prone. Hepatic lipidosis is a feature of pituitary neoplasia with hirsutism, laminitis and high plasma cortisol.

The liver is able to regenerate, and the objective in cases of acute liver failure is to provide supportive care until its function is restored. Depending on the cause and development, SDH and AST liver enzymes are at increased concentration in the blood. If biliary obstruction occurs (chronic cases) GGT and ALP are increased and conjugated bilirubin exceeds 25% of the total. In acute cases oral, or i.v., glucose administration is frequently helpful and imbalances in electrolyte, acid–base and hydration status should be gradually rectified. In cases of hypoglycaemia, 10% glucose i.v. should be administered to establish normoglycaemia and then 5% glucose, at the rate of 2 ml/kg/hour, given for the initial 24 hours. Blood glucose should be monitored and the post-absorptive value maintained approximately between 4.5 and 6 mmol/l plasma. Hyperlipaemia may be moderated by oral dosing with methyl donors such as choline chloride, or betaine and a general B-vitamin supplement. If signs of hepatoencephalopathy occur, a reduction in the absorption of toxic metabolites from the gut is aided by giving mineral oil (contraindicated in horses with GI reflux) carefully by nasogastric tube, as a laxative, at the rate of 2–4 l/500 kg BW once daily. Horses should be kept out of intense sunlight, to reduce the risk of photosensitization. High protein diets, legume hays and haylage, or silage, should be avoided. A mash, mixed with water, of the composition shown in

Table 11.5 Clinical signs and abnormalities of liver disease (West 1996). (Note: characteristics within rows, i.e. signs and clinical abnormalities, are not necessarily related.)

Signs	Clinical abnormalities
Weight loss	Increased serum glutamate dehydrogenase (EC 1.4.1.3)
Anorexia	Increased protein and neutrophil counts in abdominal paracentesis samples
Dullness and depression	Intravascular haemolysis and haemoglobinuria
Jaundice	Raised haematocrit, owing to dehydration
Tachycardia	Leucocytosis, owing to neutrophilia
Intermittent pyrexia	Elevated serum bile acids ¹
Abdominal pain	High serum GGT (EC 2.3.2.2) in biliary-tract damage ²
Ventral oedema	Elevated plasma ammonia ²
Muscle fasciculations	Increased prothrombin time ³
Diarrhoea or constipation	Low plasma fibrinogen and platelet count
Dysphagia	Frequently normal values of:
Photosensitization	• plasma urea ⁴ , but may be low, or high in renal failure
Encephalopathy	• plasma glucose, but severe hypoglycaemia terminally
Mucosal petechiation and nasal bleeding	• hyperbilirubinaemia ⁵

¹ Serum bile acids are sensitive measures of early *S. jacobaea* toxicity and with glutamate dehydrogenase, GGT and liver biopsy are useful in diagnosing different types of liver disease.

² May be raised in both intra- and extrahepatic cholestasis.

³ Delayed coagulation is a definite contraindication for liver biopsy, as haemorrhage into the abdominal cavity is likely to result.

⁴ Normal ammonia:urea ratio ($\mu\text{mol}:\text{mmol}$) is 3:1. A ratio of 40:1 carries a poor prognosis.

⁵ Frequently with acute hepatic failure. High concentrations are a terminal change. The conjugated form rarely exceeds 25% as the equine kidney is only permeable to conjugated pigment. A conjugated fraction of greater than 35% is normally terminal, whereas high total bilirubin, when most is unconjugated, may follow a few days of starvation.

Table 11.6 Composition of wet mash (g/kg diet), 4–6 times daily, for acute liver disease.

Crushed barley	326
Crushed oats	400
Beet pulp	150
Beet molasses	120
Lysine-HCl	2
Vitamins* plus trace elements	0.5
Limestone	1.5
Grass hay	1 kg maximum

*Should include choline chloride, 1 g daily.

Table 11.6 and allowed to stand for 30 min before feeding is recommended. Anorectic horses can be force-fed a gruel by nasogastric tube.

Thiamin and folic acid may be given parenterally once per week. There may be a case for raised, or pharmaceutical, doses of niacin to reduce adipose tissue lipolysis and FFA flux (see 'Hyperlipaemia', this chapter). Some benefit may be obtained by

Table 11.7 Amino acid supplement (g/500kg BW daily) for anorectic horses.

L-leucine	6
L-isoleucine	4
L-valine	5

including synthetic sources of the branched-chain amino acids leucine, isoleucine and valine in the gruel (Table 11.7).

CHRONIC WEIGHT LOSS

Apart from the effects of an inadequate diet and bullying, other causes of weight loss are many, but GI diseases are among the most common. There are numerous recognized and putative causes of small-intestinal malabsorption, including inflammatory bowel disease (possibly an immunological response to chronic antigenic stimulation), alimentary lymphosarcoma, idiopathic villus atrophy, verminous arteritis, intestinal ischaemia, mucosal biochemical abnormalities, mucosal oedema and lymphangiectasia (dilatation of the intestinal lymphatic system, characterized by protein-losing enteropathy). Weight loss is the predominant clinical sign, owing to inappetence, maldigestion, small-intestinal malabsorption, enteric protein loss and cachexia. Haematological evidence is normally limited to anaemia and or neutrophilia, a response to inflammation. Hypoproteinaemia, hypoalbuminaemia and a marked increase in plasma alkaline phosphatase activity were noted by Chandler *et al.* (2000). In protein-losing enteropathy, all forms of serum proteins are lost into the intestines, but the effect is greatest for plasma albumin and γ -globulin concentrations. An oral glucose absorption test is useful in measuring absorptive function, with resting and 120 min post-administration blood glucose measurements. The 120 min value should be double the resting value.

At feeding time, the activity of the horse should be closely observed. If its behaviour is atypical, the mouth should be examined and the teeth checked. Primary disorders of cheek teeth seem to be dominant. These include abnormalities of wear, traumatic damage, infections and fractures. Nasal discharge is more commonly present with caudal than with rostral (adjacent to the muzzle) maxillary cheek teeth infections. The response to treatment is good (Dixon *et al.* 2000a & b).

If the buccal cavity is normal, the problem may lie in other regions of the GI tract. Increased energy expenditure can result from chronic infections, neoplasia, or chronic obstructive pulmonary disease (COPD) (see this chapter). Therapy for most causes includes correction of fluid deficits, hypoglycaemia and acid–base imbalances and the provision of a high-quality diet given in small amounts at frequent intervals, supplemented with micronutrients. Stable design is important, as for example Fuller

et al. (1998, 2001) made the interesting observation that reduced daylight length led to a decrease in feed intake five to eight weeks later (see Chapter 7).

One of the following causes might be suspected in cases of chronic weight loss (protein-losing syndromes are discussed above in 'Protein-losing gastroenteropathy' and 'Protein-losing nephropathy'):

- jaw and dental abnormalities, including sharp points on the upper and lower molars and premolars, or abscesses below the teeth;
- roundworm infestation damaging the gut, which has interrupted the uptake of nutrients, and larval stages injuring mesenteric blood vessels that supply the intestines;
- diarrhoea;
- tuberculosis which very occasionally involves the digestive system;
- liver disease or the presence of some chronic septic focus in the body;
- windsucking;
- shy horses, low in the social order and subjected to group feeding;
- heart abnormalities and anaemia;
- arthritis and other causes of chronic low-grade pain; and
- cancer, especially in older horses. Grey horses are prone to internal melanomas.

THE MATURE SICK OR GERIATRIC HORSE

The following points are a guide to the nutritional management of the adult sick horse:

- Provide fresh clean pasture which is sheltered and within observation distance;
- Where concentrate feeds form a major portion of the diet remember that it is necessary primarily to meet the energy requirement. To this end those feeds offered should be edible without difficulty. Grains should be crushed and made palatable with a little molasses. The horse is likely to be fussy and so a range of feeds should be offered to find those most acceptable;
- Include readily consumed bulky treats, such as apples and sliced mould-free carrots;
- Vegetable oil is a useful addition to the feed as it will not cause colic;
- Give B vitamins, including vitamin B₁₂ by injection;
- If digestive function is compromised, partial parenteral nutrition (PN) is useful to reduce the load on the GI tract. Total PN may be adopted for a few days. This should be introduced, and subsequently removed, gradually over several days. Where maintenance of fluid status is necessary, acid-base status should be assessed and Ringer's, or lactated Ringer's, solution should be given i.v. through a separate catheter.

Many of these points may apply to the old horse. Teeth are likely to be in poorer condition than in younger animals and therefore small quantities of high-quality

leafy forage will be required to supplement larger quantities of digestible concentrates. These can usefully include micronized cereals. Ralston & Breuer (1996) reported that a commercial feed containing 85g protein, 2.7g Ca and 2.2g P/kg was inadequate when it represented 20%–100% of the total feed, with the remainder being timothy and lucerne hay. A compound feed containing 140g protein, 6g Ca and 4–6g P, given in similar proportions with the hay and providing similar amounts of DE, led to greater body weight gains, better condition, higher plasma total protein and P concentrations, and higher haematocrit and blood haemoglobin values. Renal function was not compromised by the larger protein intake, as assessed by plasma creatinine concentration. Where older horses are known to have poor renal function, the dietary protein quality, i.e. amino acid balance, should be ideal and the dietary protein allowance should prevent any excessive rise in plasma urea level.

MUSCLE AILMENTS

There are several metabolic abnormalities connected with muscle ailments, for which many of the signs are shared and attached to which the nomenclature is frequently confused. Previous terms include ‘tying-up’, myositis, set-fast and azoturia.

Exercise-associated myopathy

There is a range of degrees of exercise-associated myopathy, from severe rhabdomyolysis with azoturia, to milder ‘tying-up’, although the terminology is variable. The onset of clinical signs of muscle disease usually occurs within 5–20 min of commencing exercise which may be either mild or strenuous. Signs after racing are also seen in racehorses. Muscle damage is accompanied by an elevation in serum CK and AST.

Post-exhaustion syndrome

Muscle problems occur some time after severe, prolonged exercise which causes exhaustion. Signs may be presented two to four days after completion of the exercise. Damage may involve both skeletal and cardiac myopathy, hepatic lipidosis, renal damage, laminitis and GI ulceration. The onset of muscular malfunction may occur within a few initial strides, when the horse falters and stops, generally refusing to move. The muscles are not palpably abnormal, but are stiff and sore. The horse may adopt a cramped stance and, if willing to move, it does so reluctantly and slowly. Lesser attacks may occur following cooling off. Muscular stiffness, which passes within one to three days, is associated with the muscular accumulation of lactate. The activity of serum CK returns to normal after four to six days, but that of

AST may take four to five weeks to do so. In addition to a depletion of muscle glycogen (principally in the fast-twitch fibres), there is also a depletion of ATP and CP. However, muscle glucose as well as lactate concentration is raised, suggesting local hypoxia (insufficient oxygen reaching the muscle cells from the blood). A leakage of the muscle enzymes AST, CK and LDH is the cause of their raised blood concentration (NB: Determination of iso-enzyme concentrations may help locate the origin, although, for example, LDH₅ is present in both liver and locomotor muscles).

The horse should be kept warm. Effective treatment normally includes the i.v. administration of calcium gluconate, magnesium and phosphate ions and vitamin D. Prevention requires training to increase condition, more frequent rest periods, and the administration of electrolyte solutions during and after physical activity. There is, moreover, some evidence that 'tying-up' is associated with elevated phosphate clearance and that feeding calcium carbonate is helpful. The condition is also occasionally noticed in fillies coming into season (oestrogens increase the activity of 1-hydroxycholecalciferase, which further implicates vitamin D).

After hard work it is an advantage to trot or canter slowly as this will stimulate the transport of lactic acid from the muscles to the liver in healthy horses so that muscle and blood pH return to normal more rapidly. Moreover, this light exercise stimulates the flow of oxygen to the muscles, accelerating the conversion of lactic acid back to glycogen. Accordingly the static changes are avoided.

Equine exertional rhabdomyolysis syndrome (ERS)

Exertional rhabdomyolysis (azoturia, tying-up, set-fast) primarily affects musculature and occurs in most breeds and ages of horse. Its clinical differentiation from intoxication by ionophores, aortic-iliac thrombosis, atypical myoglobinuria, and exhaustion, following protracted exertion, requires careful analysis of clinical signs and laboratory determinations (P.A. Harris personal communication, 2002). The clinical signs tend to occur during or after exercise, but also in horses at rest, or after they leave their box or field. They are presented as a slight shortening of stride to a complete inability to move and recumbency. The condition can be fatal. There is a fall in muscle pH and muscles tend to appear firm. Palpation may not be resented. Sweating is excessive and elevated pulse and respiration rates are evident. Discoloured urine may be voided in severe cases, although this is also seen, for example, in monensin (see Chapter 5) and acorn poisoning. Myoglobin is usually present in the urine when its plasma concentration exceeds 0.2 g/l and this can lead to nephrosis and uraemia. Simple diagnostic methods used to distinguish myoglobin from any urinary haemoglobin contain inaccuracies. The muscle enzyme CK exceeding 200 iu/l attains a peak concentration in the blood 6–12 hours after a single severe episode of muscle damage and returns to normal frequently within a week, if no further damage occurs. AST reaches a peak about 24 hours after the episode, but normal values are not regained for two to four weeks. Very high plasma concentra-

tions of CK and raised AST are also observed following strenuous exercise, although they may not represent muscle damage and normal values may be achieved after 24 hours.

The aetiology of ERS is still a mystery. It may indicate poor management of training and it commonly occurs within the first hour of exercise. The ailment usually does not follow a rest period of only one day, or rest as long as 14 days, but commonly occurs following two days' rest on full rations. Some forms of the disease that succeed extended exercise may be related to a depletion of glycogen stores and an increased shift to mitochondrial β -oxidation. An interruption in regular exercise, with the maintenance of an immoderate dietary starch intake, seem to be frequently associated factors.

Horses may be in a state of dehydration and metabolic alkalosis, or acidosis, and therefore solutions of sodium bicarbonate should not be given unless the acid-base and electrolyte status (see 'Laminitis', this chapter, for K status) has been established. In the absence of this knowledge, isotonic neutral solutions, such as Ringer's, should be given i.v. Diuresis should be induced (and anyway would do no harm) to diminish the risk of nephrotoxic effects of myoglobinuria. It is vital that the horse is not allowed to move at all, as recovery requires that a regime of complete rest is immediately instituted. It may remain on its feet, or become completely recumbent, and severe pain and distress are often accompanied by repeated attempts to rise. The horse should be removed to its stable as quickly as possible in a low-loading trailer, where every effort should be made to keep it standing by slinging, or other means, as this may prevent the development of uraemia. Not only racehorses but also draught horses on heavy cereal rations and those kept on lush pastures during the week and ridden only at weekends may be susceptible.

Diets differing in DCAB value have similar effects on both ERS and healthy horses, as measured by plasma electrolyte concentrations, CK activity, plasma and urine pH and renal fractional excretion values of Na, K, Cl and P (McKenzie *et al.* 2002). Yet myoplasmic concentrations of free Ca increase in ERS, associated with damage to mitochondria and other organelles. Dantrolene sodium, a skeletal muscle relaxant that acts primarily by affecting Ca flux across the sarcoplasmic reticulum, lessened the elevation of post-exercise serum CK levels and appeared to eliminate signs of ERS amongst 77 TBs (Edwards *et al.* 2003). Thus, a breakdown in normal intracellular Ca transport seems to be a critical cause.

One study (Valberg *et al.* 1993) showed that horses suffering from recurrent ERS had a higher ratio of type IIA to type IIB muscle fibres, but this may have reflected better training which caused a lower accumulation of lactate and ammonia during near-maximal exercise (see also 'Ammonia toxicity', this chapter). Blood cortisol and glucose concentrations tend to be higher in ERS horses, although this may be an effect of the stress, rather than a cause of the ailment.

If exercise and movement have been stopped and treatment instituted immediately the horse may recover in two to four days. Treatment has included narcotic drugs and corticosteroids administered i.v. to control the swelling and to stimulate energy metabolism, and i.v. or oral administration of electrolytes in large quantities

to maintain a high rate of urine flow at an alkaline pH, preventing myoglobin precipitation in the renal tubules. Although acidosis is a normal feature of azoturia this should not be assumed without analysis of plasma acid–base and electrolyte status. Some horses have been found to be alkalotic, when, of course, treatment with sodium bicarbonate would be harmful. Intramuscular injections of 0.5 g thiamin repeated daily seem to be warranted, and the inclusion of pantothenic acid and riboflavin, also involved in oxidative energy metabolism, may be justified. If the painful swelling of the muscles is not reduced, pressure on the sciatic nerve can induce secondary degeneration of other muscles. The maintenance of proper kidney function is vital if health is to be restored.

ERS of polo horses is said to occur more frequently early in the season in those that are excitable and unfit (McGowan *et al.* 2002). The risk of ERS is reduced if horses are always warmed up slowly, their concentrate allowance is halved at weekends, or at other times when they are not worked and their work is reinstated gradually with a gradual increase in the consumption of starchy and high-protein feeds. The addition of dimethylglycine to the feed, as a prophylactic measure, has been recommended, but without any convincing supportive published evidence. The thyroid hormones T_4 and T_3 are intimately involved in resting energy metabolism and it has been suggested that transient hypothyroidism may occur in ERS, so that nutritional supplementation with thyroxine may assist recovery.

Polysaccharide storage myopathy (PSSM), resulting in glycogen accumulation, is a cause of exertional rhabdomyolysis of Quarter Horses and in recent years has been identified in many breeds. Clinically, stiffness, weakness and atrophy, especially of the rump, thigh and back muscles is apparent. PSSM can cause muscle glycogen levels at rest to be 2.4 times normal. The cause is increased glycogen synthesis rather than decreased utilization, so that horses with exertional rhabdomyolysis may benefit from a diet low in soluble carbohydrate with fat added as exercise rate increases (Valberg *et al.* 1999). Valentine (2003) reported that effective treatment required the elimination of grains, yielding a high-fibre diet containing by weight less than 33% starch and sugar, and the addition of 0.5–1.0 kg of a 20% rice bran fat, so that 0.45 kg fat/450 kg BW was given daily.

Recurrent exertional rhabdomyolysis (RER)

Horses in training with recurrent exertional rhabdomyolysis have moderately elevated AST and/or CK concentrations in blood samples taken at rest, even if they have been free of clinical signs for weeks. There appear to be subclinical episodes of rhabdomyolysis after exercise. Muscle-fibre necrosis and associated increases in plasma AST, CK and myoglobin occur with exercise more frequently than can be detected clinically (Valberg *et al.* 1993).

Infectious myopathies

Myopathy may result from bacterial, viral or parasitic infection.

Hyperkalaemic periodic paralysis

(See also 'Potassium', Chapter 3.) Recurrent muscle cramping, fasciculations and weakness are associated with hyperkalaemia, which is a disease for which horses have a genetic predisposition. The condition is confirmed by oral potassium challenge. There is debate as to whether or not this is associated with a breakdown in normal post-exercise potassium uptake into skeletal muscle under β_2 -adrenoreceptor stimulation by epinephrine.

Nutritional myopathy

Horses with nutritional myopathy present with depression, weakness, dysphagia and dropping of the head and neck (see also 'Selenium', Chapter 3 and 'Vitamin E', Chapter 4, especially for prevention of EDM and EMD). Muscle enzymes are elevated in serum and there is severe degenerative myopathy with hyalinization and fragmentation of muscle cells. The condition occurs typically in neonatal foals, but also in adult horses, and is associated with deficiencies of selenium and/or vitamin E.

Hypo- and hypercalcaemia and stress tetany

Extended exertion, particularly in hot weather, leads to dehydration, loss of electrolytes and to energy depletion. The signs of fatigue presented may reflect a combination of these losses even though the diet is quite satisfactory. Ca losses in the sweat can amount to 350–500 mg Ca/hour, and continued hyperventilation is associated with alkalosis (discussed in Chapter 9). An acute life threat in horses suffering from stress tetany may be posed by hypocalcaemia, in which total plasma levels can fall to 1.5 mmol/l, whereupon muscular twitching and cramps are manifested. The fall is largely the result of alkalaemia. Occasionally hypomagnesaemia may be present. On the other hand, Standardbred trotters subjected to high-speed tests exhibited a decrease in plasma ionized Ca^{2+} concentration, despite a fall in blood pH and a rise in plasma lactate, intact parathyroid hormone (PTH) and inorganic phosphorus concentrations (see 'calcium and phosphorus', Chapter 3); whereas low-speed exercise resulted in an increase in plasma pH, but with no change in lactate, ionized Ca^{2+} , total Ca, inorganic P or intact parathyroid hormone concentrations (Vervuert *et al.* 2002). The authors suggested that intact PTH is a mediator in counter-regulation of exercise-induced hypocalcaemia.

As normal muscle function, including that of cardiac muscle, requires the concentration of Ca in the blood to remain within strict limits, the most immediate need is for careful i.v. administration of a Ca gluconate solution (Table 9.3, p. 328) while heart function is monitored, as an excessive rate of administration can be fatal. Where the clinical signs of hypocalcaemia are clear, a greater risk is entailed in awaiting confirmation by laboratory determination so that immediate veterinary treatment of this kind is indicated.

A fall in plasma Ca and of Mg also occasionally occurs in lactating mares, although clinical signs are quite rare (see Chapter 7). Where tetany does occur this is usually precipitated by additional stresses of exertion, transport, weather or disease. Ca gluconate should be given. In the dairy cow hypocalcaemia causes paresis rather than tetany.

Electrolyte losses in extended exercise

Several days' rest are required for the regeneration of muscle-cell glycogen reserves after extended work. The exhaustion of this and loss of cellular potassium in sweat contribute to a sense of fatigue and, as suggested in Chapter 9, recovery during the next few days may be accelerated by providing potassium chloride as well as common salt in the feed (10g NaCl plus 5g KCl/kg total feed is recommended). Isotonic dehydration depresses thirst so that rehydration is also stimulated by the provision of these electrolytes. Salt licks are supplied in most horse boxes, but many horses will not consume sufficient in this form and the licks normally contain sodium chloride only. Thus, a powdered feed supplement containing both salts, available from feed merchants, yields a more satisfactory outcome. In fact, during summer weather generally, many horses in training for flat races do not consume enough salt from licks and therefore present a higher PCV and plasma viscosity than should be the case. By encouraging water consumption, electrolytes can have the effect of improving performance. Similarly, during long endurance events, those horses that do not drink fatigue more easily and are less likely to finish. Thus, by satisfying a need for electrolytes at rest points, a thirst response is induced, water consumption is increased and dehydration is deferred or prevented.

Losses of calcium, chloride and potassium throughout long rides may cause 'thumps' or synchronous diaphragmatic flutter during or after exercise. Losses of these electrolytes and 'thumps' may also be brought on by severe diarrhoea. A decrease in the plasma concentration of calcium, chloride and potassium is thought to change nerve irritability, initiating a contraction of the diaphragm muscles in unison with that of the heart beat. This is seen as sudden movements of the horse's flanks. Treatment consists of replacing lost electrolytes, which are always beneficial and never detrimental as long as water is available. The quantities to be administered are given in Chapter 9. The exhausted endurance horse is usually alkalotic, contributing to the hypocalcaemia, so that Ringer's solution, which is slightly acid, is preferred for immediate use. Sodium bicarbonate should not be used in these circumstances unless metabolic acidosis has been demonstrated.

LAMENESS

In one survey of 314 TBs, 53% suffered lameness at some time and in 20% of the cases lameness prevented subsequent racing (Jeffcott *et al.* 1982b). Undoubtedly,

the condition represents a considerable embarrassment to the industry and is a problem in horses and ponies of all types.

Lameness can be defined as a disturbance of gait, which reduces the weight on the affected limb. Although there are a multitude of causes, one study carried out by the author (unpublished observations) on horses in the Far East revealed that faulty mineral nutrition, as estimated by phosphate clearance, all too frequently was associated with vertebral fractures, and probably with fractures of other kinds. Unsoundness of joints may result from sprains, strains and jarring forces causing inflammation, which may also result from osteoarthritis. Although this and many bone disorders, for example, splints, ringbone, osselets, bone spavin, curb, capped hocks and thoroughpin, are unlikely to have specific dietary causes, the severity of response may well be affected by dietary imbalances or deficiencies (see 'Polyunsaturated fatty acids', Chapter 5). Abnormalities in mineral, trace-element and vitamin nutrition are frequently associated with various types of lameness, but little research work (apart from investigations of OCD, see Chapter 8) has been undertaken to make any objective assessment of the scale of that involvement. Other reasons for lameness include bruised feet, bowed tendons, navicular disease and spinal lesions, which may in part implicate poor hoof care: inspection of hooves daily may often avoid long-term problems.

Phyinitis has already been discussed in Chapter 7 and some reference was made to contracted tendons (Plate 7.1, p. 261). The latter often occurs in foals that are doing well with mares having ample milk on good grass. The speed of onset in a foal is surprising, in that within 24–48 hours the heel will rise and a slight concavity develop on the front wall of the hoof. Wear at the heel is decreased and increased tension occurs in the extensor tendons. These do not contract, but apparently fail to develop at a rate commensurate with bone growth, and the fetlock joint also tends to enlarge. It is essential to spot the aberration in the early stages so that it can be counteracted by weekly rasping of the heels, exercise, the removal of concentrates and reduction in milk intake. Ultimately, surgery may be the only means of rectifying severe angular deformities of leg joints (see also 'Developmental orthopaedic disease', Chapter 8). Exercise is important in the prevention of leg-growth abnormalities, which may be more prevalent where mares and foals spend long periods in their boxes during adverse weather without any cut in feed intake. It may be preferable to allow mares and foals to remain out all the time in the summer, regardless of weather, so long as some form of shelter is available.

Navicular disease

Lameness and damage to the navicular bone of the hoof are possibly caused by thrombosis of the navicular arteries, abnormal stresses on the bone or by degenerative changes. Treatment includes corrective trimming of the hoof and fitting a wide, webbed egg-bar shoe. Drug treatment with isoxsuprine hydrochloride, a vasodilator, is a common practice, as it avoids the risk of haemorrhage encountered with warfarin. Warfarin (dicoumarol) may be helpful, although its use is declining. This

drug interrupts blood coagulation by extending prothrombin time. The dose has to be carefully titrated to extend clotting time by 2–4s from the equine standard of 14s. An excessive dose will lead to bleeding. If colic inadvertently occurs during treatment, an extended prothrombin time resulting from depressed liver function requires stopping warfarin treatment and starting vitamin-K treatment.

It is essential that horses treated with warfarin are fed consistently, particularly in respect of the amount of green feed, which is rich in vitamin K, and as the level of work affects clotting time any change in activity must be imposed gradually. If practical, regular work should be begun and blood samples taken at intervals of at least one per month during rest periods, but immediately after exercise. Samples should also be taken five to seven days after any change in work or feed routine. Veterinary treatment with warfarin must always err on the side of caution as excess can be fatal. The drug seems to act by reducing the viscosity and increasing the flow of blood, which improves the nutrition of the navicular bone, and this is probably more than just the prevention of thrombus formation.

Osteoclastic activity also plays a role in the pathology of navicular disease. The bone remodelling changes that occur may be corrected by tiludronate, a bisphosphonate, that inhibits bone resorption. When given i.v. at the rate of 1 mg/kg BW daily for ten days, tiludronate improved lameness two and six months post-treatment, so long as treatment was commenced soon after the first clinical signs were observed (Denoix *et al.* 2003).

HOUSING

Any detailed consideration of housing is beyond the scope of this book, but the environment provided by housing has a profound impact on the well-being of horses. Aberrant behaviour, including wood chewing, wall licking and kicking, or pawing by Quarter Horse weanlings was seen to be much more frequent amongst those box stalled than those weaned in a paddock (Heleski *et al.* 1999). A series of studies by Houpt & Houpt (1988) at Cornell University, Ithaca, New York has revealed environmental factors important to the well-being of horses. For example, mares in visual contact with other mares are less active and spend more time eating than do those without visual contact. Mares were also found to prefer an artificially lit environment to a dark one. Environmental temperature studies with *ad-libitum*-fed weanling horses (Cymbaluk *et al.* 1989a) showed that feed intake increased by 0.2% for each 1°C decrease in barn temperature below 0°C. Enrichment of the stable environment through provision of multiple forages was also shown to reduce straw consumption and motivate foraging behaviour (Goodwin *et al.* 2002).

Ventilation

For reasons of savings in finance and labour there is a trend for housing racehorses in American-style barns (Townson 1992). Less attention to the detail of ventilation

is required in the construction of individual horse loose boxes. On the other hand, the communal air and the aerial dust and ammonia levels that can develop at feeding and bedding-down times in barns dictate strict rules that should be followed for ventilation rates, as this is the principal route for evacuating the small particles that are the agents of respiratory distress.

In recommending ventilation rates, allowance has to be made for the fact that air volume per horse in barns tends to be more than double that in loose boxes (mean values 98 cf. 43 m³/horse; Townson 1992). Natural systems of air flow (Fig. 11.2) should have a controllable inlet area of up to 0.3 m² per horse (Sainsbury 1981). If building design impedes natural air flow, fan assistance should be provided at the base of the outlet chimney. This will function as an aid to natural ventilation and is a more desirable solution than the installation of a pressurized system for which the costs are greater and the numbers of suspended air particles increased by greater air turbulence. There should, for similar reasons, be no recirculation of air as occurs in air-conditioned units, as seen in hot climates, where insulation, especially of the roof, is critical.

Abrupt changes in air flow as a result of 'on-off' regulators are to be avoided. Bottom-hinged air inlets are recommended, which deflect cold air up in cold weather and may be fully open in hot for cross-draughts. Their height above the floor should be such as not to interfere with the stock and they must, of course, be of safe construction. Additional inlets will probably be necessary for very hot weather. Where horses are grouped in barns and covered yards a copious air flow is essential at all times. This will be facilitated by a wide open ridge, 0.3–0.6 m wide, with a covering flap. Space boards at the top half of the wall are successful (150 mm wide boarding with 25 mm gaps; Fig. 11.2) or, on exposed sites, narrow sliding boards in which one set slides over the other.

The air space in barns is shared, whereas that in loose boxes is either shared or isolated. Although it should be isolated, the tendency is for sharing to be more frequent in modern loose boxes. Probably the most successful housing in the UK consists of various forms of monopitch lean-to buildings (Figs 11.3 and 11.4), which contain single open-fronted boxes facing the warmest wind and the greatest sunlight. These buildings have a low back and high front with hopper-flap air inlets such that the air flows from back to front. An extension to the roof at the front gives cover and cross-partitions act as load bearers. These divisions should be solid as the partly open ones allow crossflow of air, the incidence of wall kicking increases and, where the division contains iron bars, even at quite high levels, shod horses can become ensnared (see also 'Ailments related to housing', this chapter).

Dust loads vary considerably, not only among stables, but also within a stable at various times in the routines of daily husbandry. The horse should be out of the environment when conditions are at their worst and ventilation should be encouraged at that time for the safety of the grooms. Dust can contain pathogens, allergens, irritants and nonpathogenic nuisance microbes. Townson (1992) found that 65% of racehorses in Ireland were bedded on straw, and all were given hay as forage. When this is provided in large round bales, and shaken out in the feed passage before

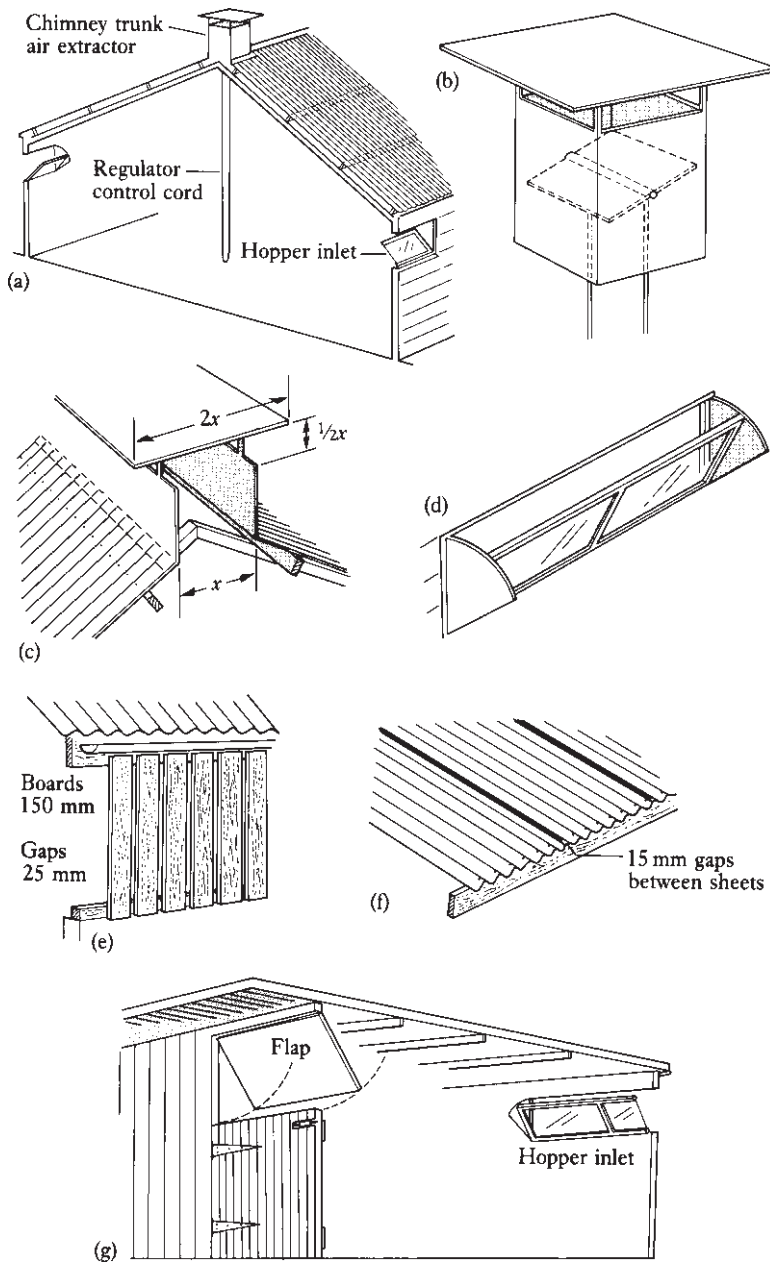


Fig. 11.2 Arrangements for the natural ventilation of stables: (a) ventilation suitable for all stables using extractor chimney trunks and hopper inlets for fresh air; (b) detail of the extractor chimney trunk, which may have a regulator or electric fan placed in the base; (c) simple open ridge suitable for extraction ventilation of covered yards; x is normally about 300 mm in yards up to a width of 13 m and 600 mm in yards over 13 m and up to 25 m wide; (d) hopper window suitable as a fresh-air inlet; (e) spaced boards, giving draught-free ventilation: normally 25 mm gaps between 150 mm wide boarding; (f) 'breathing roof' – corrugated roof sheets fixed with 15 mm gaps between for extractor ventilation; (g) mono-pitch house showing hopper inlet at back and ventilating flap at front. Note overhang on roof to protect horses from rain, sun and wind (Sainsbury 1981).

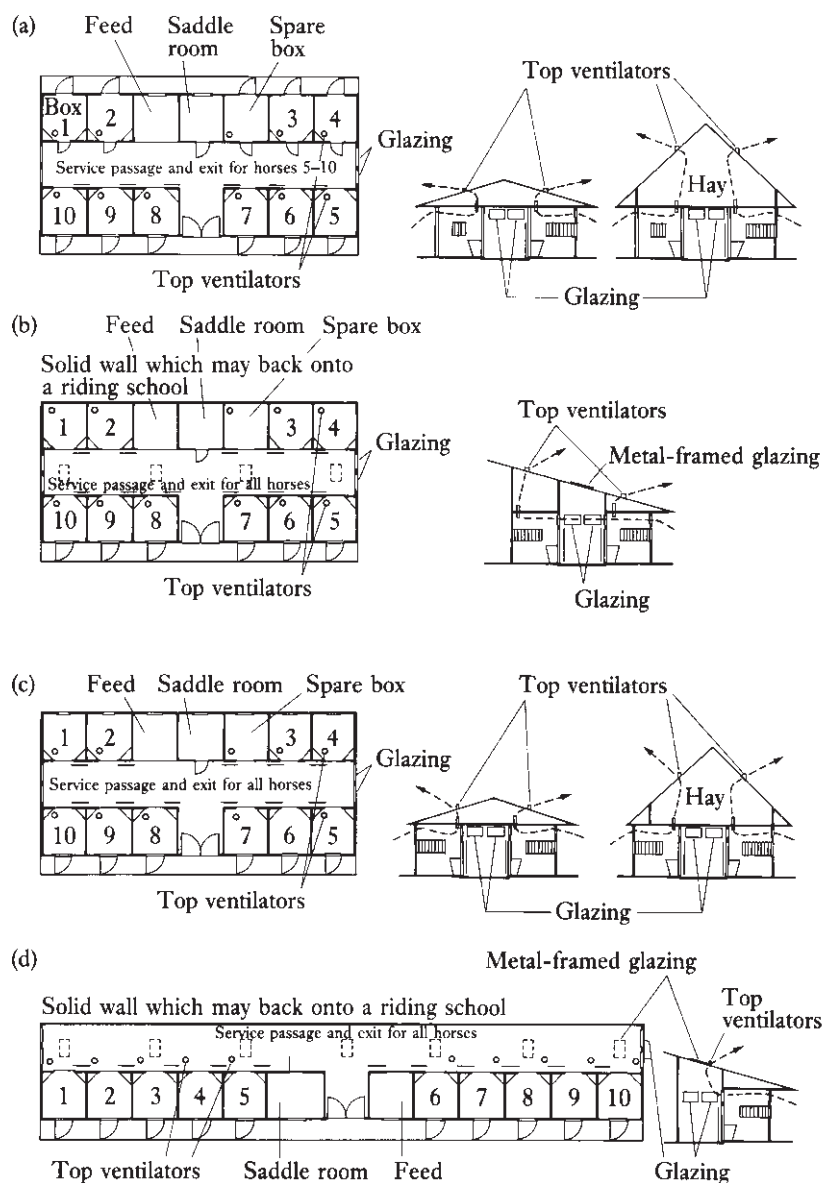


Fig. 11.3 Four layouts of ten horse boxes, which combine the advantages of indoor and outdoor boxes without draughts. (a) All the boxes open to the outside: boxes 1-4 have large exterior service doors; boxes 5-10 have interior sliding doors and shutters opening to the outside. (b) Stable with a solid back wall and interior service; boxes 5-10 have shutters opening to the outside. (c) Stable with a side exposed to bad weather and with interior service; boxes 5-10 have shutters opening to the outside. (d) Stable with a solid back wall and with interior service; all boxes have shutters opening to the outside (Ministère de l'Agriculture 1980).

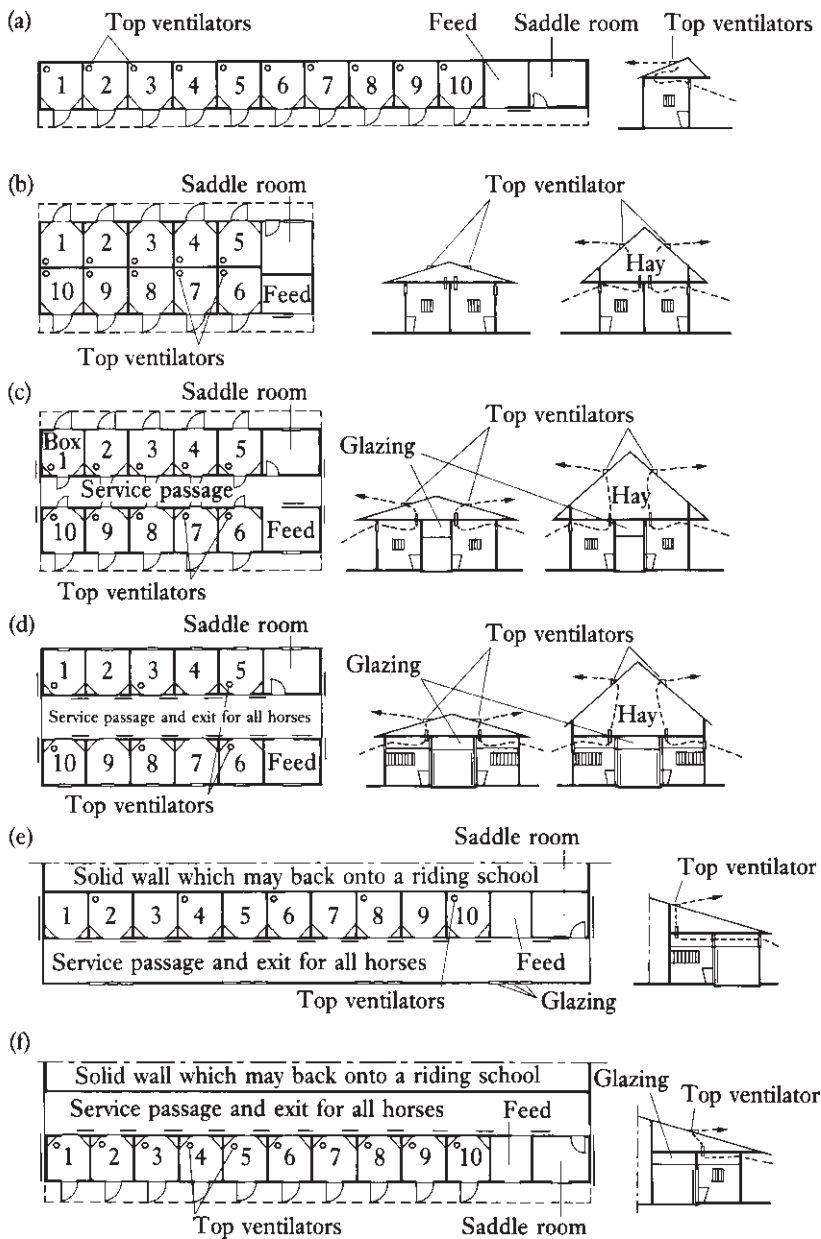


Fig. 11.4 Simple layouts for ten horse boxes: (a) a row of outside boxes with external services; (b) back-to-back boxes with external services; (c) boxes with external horse access and internal service; solid doors and walls in the passage keep draughts to the minimum; (d) boxes with internal (central) servicing without access for the horses' heads to the outside (a less satisfactory arrangement); (e) boxes with internal (lateral) servicing, without access for the horses' heads to the outside (a less satisfactory arrangement); (f) boxes with internal (lateral) servicing and with access for the horses' heads to the outside; solid doors and walls onto the passage keep draughts to the minimum (Ministère de l'Agriculture 1980).

Table 11.8 Number of dust particles/cm³ stable air in well-ventilated boxes (Webster *et al.* 1987) and mean weight of dust/m³ air in 1166 stables of 26 Irish racing establishments (Townson 1992).

	Number of airborne particles (diameter >0.5 µm/cm ³ air)*		Weight of dust (mg/m ³ air)
	Quiet	Bedding down	
Wood shavings	8.8	30.5	0.19
Paper	10.0	40.0	0.37
Straw	11.7	75.9	0.57

*Particles of up to 5 µm diameter may enter the alveoli of the lungs. These can include antigenic *Aspergillus fumigatus* and *Micropolyspora faeni*.

Table 11.9 Ventilation rates required to achieve a TLV of 33 particles/cm³ air at three rates of release of dust into the stable air (Townson 1992).

Dust release rates (particles/cm ³ /hour)	Air changes required/hour
60	3
300	10
600	22

feeding, it adds dust to the barn atmosphere. Where alternatives to hay are not available the hay should be of very top quality, or soaked prior to distribution. Clarke (1987) showed that the number of breathable particles released from moulded rye grass hay was reduced from 45 000 to 1650/mg fresh material after five minutes of soaking and to 525/mg after 24 hours of soaking (cf. 44/mg from haylage). Wood shavings should be used as bedding in barns, as they were found to release the lowest amount of dust of the sources compared (Table 11.8).

Too frequently an adequate ventilation rate depends on whether or not the top half of a loose-box door is open and whether or not barn doors are open. The problem tends to be greater with barns, as the top halves of loose-box doors are normally left ajar, but should have the facility for not being fully closed. The need for good ventilation should be gauged against the evidence that the thermoneutral zone for a mature and fed Quarter Horse is -10 to +10°C. Loss of body heat through concrete floors by conduction, when a horse is lying down, is greatly reduced by bedding, which also reduces floor draughts. Generally, ventilation rates should be four to eight air changes an hour. These rates should keep the number of aerial particles of >0.5 µm diameter to less than 33 particles/cm³ (the threshold limiting value, TLV). However, this conclusion depends on the release rate of dust (Table 11.9).

Table 11.10 Requirements for natural ventilation of a typical box and barn (Sainsbury 1981; Webster *et al.* 1987; Townson 1992).

Dimensions per horse	Box	Barn
Volume (m ³)	50	85
Surface area of building* (m ²)	41	43
Ventilation inlet area (m ²)	0.34	0.46
Ventilation outlet area (m ²)	0.17	0.23
Height from inlets to outlets (m)	1	2
Ventilation rate (m ³ /s)	0.055	0.094
Air movement (m/s)	0.15–0.5	0.15–0.5
Ventilation heat loss (W/°C)	67	114
Ambient temperature (°C)	0–30	0–30
Relative humidity (%)	30–70	30–70

* Assuming 10 horses in a row of boxes or single barn. W, water vapour (kg).

Recommended dimensions for natural ventilation in boxes and barns are given in Table 11.10. Natural ventilation depends on both the area of inlets and outlets and the temperature difference between inside and outside, i.e. the temperature gradient. Insulation of a stable not only increases that gradient, and therefore increases the natural ventilation rate (stack effect), for a given area of inlets and outlets, but also decreases the fluctuations in temperature of the building. Moreover, roof insulation can decrease the solar heating effect nearly tenfold compared with a single-skin roof.

Ailments related to housing

Weaving

Weaving behaviour involves repetitive lateral swaying of the head, neck, forequarters and sometimes hindquarters and is generally indicative of poor welfare. This behaviour is reduced by the visual presence of a healthy horse in a neighbouring box, or by the addition of a 1 m² acrylic mirror in the weaver's box and the presence of a hay net (Mills & Davenport 2002).

Foals

The importance of hygiene where foals are concerned cannot be overemphasized. This includes the removal of pests. Salmonellosis has been discussed already in 'Acute diarrhoea', this chapter. Severe liver damage and a lethal infection of young foals (from a few days to six weeks old) is caused by Tyzzer's bacillus (*Clostridium piliformis*). This bacterium is carried in the GI tract of many species of rodents and lagomorphs (rabbits and hares) and infection may occur by consumption of their carcasses, faeces or litter. The endospores can survive for very long periods in

rodent litter and are sensitive to heating to 80°C for 15 min, or exposure to 0.015% sodium hypochlorite, to 1% iodophol and to 5% phenol.

Heaves [chronic obstructive pulmonary disease (COPD)] and other respiratory diseases

COPD

Viruses, bacteria (*Streptococcus equi*) and lungworm cause coughing, but the most frequently encountered equine respiratory disease in the UK is chronic obstructive pulmonary disease (COPD). In a population of 300 adult horses referred for pulmonary examination in the north of the UK, Dixon *et al.* (1995) found that of 270 with pulmonary disease, 16.7% of the cases presented with infectious or post-infectious pulmonary disease, 2.6% with *Streptococcus zooepidemicus* pulmonary infection, 2.6% with lungworm infection, 5.9% with exercise-induced pulmonary haemorrhage, 3.3% with chronic idiopathic hypoxaemia, 14.1% with miscellaneous identified, or undifferentiated, pulmonary disorders and 54.8% with COPD. COPD is a reactive, inflammatory, hypersensitivity response of the lower respiratory tract, that is, the small airways (with fibrosis of the alveoli), to dust and mould (fungus and thermophilic actinomycete, especially two moulds, *Micropolypora faeni* and *Aspergillus fumigatus*). Some horses are sensitive to other organisms and even to rye grass pollen in the atmosphere of the stable, leading to a range of clinical reactions from mild exercise intolerance to severe dyspnoea, even at rest. Pirie *et al.* (2002) measured the response of heaves-affected and control horses to an inhaled hay dust suspension (HDS), principally mould spores, or to its supernatant and washed particulate fractions following centrifugation. Airway neutrophilia approached the magnitude of that found with HDS when the particulate and supernatant fractions were combined in comparison to the effect of either fraction separately. The soluble fraction seemed to be of importance in neutrophil recruitment, although HDS was important for pulmonary dysfunction. Recent evidence (McGorum 2003) that endotoxin produced by Gram-negative bacteria present in hay and bedding is a contributory cause of COPD may throw further light on the evidence of Pirie *et al.*

Breathing, in heaves, is characterized by a 'double lift', seen in the abdomen on expiration, when the normal abdominal contraction is followed by a second lift as the horse endeavours to expel more air. This recurrent airway obstruction is known as 'broken wind' or 'heaves' in which parts of the lung tissue lose their elasticity. Individuals between six and ten years old are most frequently affected.

Evidence indicates that more precipitins to mould antigens generally occur in the sera of horses housed in barns than in those in boxes. However, this does not necessarily relate to the risk or severity of, COPD. Consequently, some researchers hold the view that COPD horses may demonstrate precipitins to these fungi as a consequence of impairment to pulmonary function. The confusion may partly arise from the evidence that hypersensitivity has a rather strong genetic basis.

Sensitization and the presence of serum-precipitating antibodies to mould antigens do not necessarily lead to clinical disease in a particular horse, but when clinical signs of dyspnoea occur the IgA:albumin ratio of bronchioalveolar lavage (BAL) fluid increases and pulmonary neutrophilia of this fluid is frequently found. Relief is achieved by veterinary intervention with a cyclo-oxygenase blockade. Whether dietary fats rich in n-3 fatty acids would reduce the risk of inflammatory responses (see Chapter 5) has not to the author's knowledge been examined. Susceptible horses should be removed from an environment of high allergen loads, otherwise the forced abdominal breathing, caused by bronchospasm, excessive mucus secretion and inflammation of the airway mucosa is likely to occur during periods of raised atmospheric concentrations of allergens.

There is probably too great a concern that horses will catch a chill at night unless doors and windows are closed, and this may often lead to inadequate ventilation, which, with inappropriate bedding, is the most important environmental factor in COPD. Inflammatory airway disease is twice as likely to occur in horses bedded on straw compared with those bedded on shredded paper (Holcombe *et al.* 2001). However, horses prefer wheat-straw bedding to dust-extracted wood shavings, which are in turn preferred to shredded paper. Straw leads to more bedding-related activities, yet the welfare significance of this is unclear (Mills *et al.* 2000). Although horses prefer wheat straw, either shavings or shredded paper should be used as bedding to reduce the risk of COPD.

COPD typically presents with a chronic cough, nasal discharge and expiratory dyspnoea. The widespread endoscopic examination of airways, with cytological examination of respiratory secretions and use of other diagnostic tests has led to the conclusion that COPD is an important cause of poor performance in the absence of any other overt clinical signs. In addition, there has been the recognition, with increasing incidence, of summer-pasture-associated obstructive pulmonary disease. Many practitioners in the UK are of the opinion that the prevalence of COPD is actually increasing, although cases of lower severity may now be identified. Earlier cases were characterized by elevated maximal intrapleural pressure changes (max dPpl), whereas Dixon *et al.* (1995) found elevated max dPpl values in fewer than half of their 300 referred cases.

Both genetic and environmental factors influence the IgE response to mould allergens from *Alternaria alternata* and *Aspergillus fumigatus*, causing COPD (Eder *et al.* 2001), indicating differences in risk amongst horses. The existence of COPD in a horse predisposes it to additional bronchial hazards. McGorum *et al.* (1998) observed that the total airborne endotoxin concentration (standardised against *Escherichia coli* 0111:B4 endotoxin) in conventional stables is excessive. They suggest that the minimal total airborne endotoxin concentration causing bronchoconstriction in horses with pre-existing airway inflammation, such as occurs in COPD, may be much lower than that for normal horses. There are many similarities between COPD in horses and asthma in man (which has shown increased prevalence over the last two decades). It has been proposed that air pollu-

tion with nitric oxide and related hydrocarbon combustion products predisposes patients to respiratory disease. The mechanism probably involves the induction of airway inflammation and increased epithelial permeability, with reduced clearance of allergens and other inhaled particles that may trigger COPD development.

Other respiratory diseases

COPD occurs in individual horses and is not infectious, but there are other respiratory diseases that spread from horse to horse and cause impairment and damage to pulmonary function. The principal transmissible diseases are caused by bacteria, parasites and viruses. Viral infections seem to be of increasing incidence among horses and ponies and include equine influenza, equine herpes viruses I and II, rhinoviruses I and II and adenovirus, particularly in foals. It is thought that the widespread occurrence of viral infections may be associated with the trend to have larger numbers of horses in close proximity in totally enclosed buildings, greater national and international traffic in horses and, it must be said, an increasing awareness and understanding of viruses. Lung damage caused by these infections may leave horses more subject to the allergic responses of COPD: certainly one aggravates the other and both are influenced by building design and atmospheric pollution.

It is concluded that horse boxes should be not only well ventilated, but also sited distant from road traffic. Where haylage or damped hay is impractical to use, some benefit may be obtained from the use of mould-spore-extracted hay, or from the installation of ventilated hay racks, into which the air is drawn and extracted from the building by a fan. The system can be used as the ventilation system for the box, which should be bedded with wood shavings or paper. Prophylactic use of sodium cromoglycate has been found effective in sensitive individuals. However, prevention is better and less expensive than a partial cure.

All hay contains moulds, but some is visibly mouldy or musty and therefore presents a greater hazard than hard, stemmy, clean and shiny hay. Thus, there is reasoned justification for the use of such safe, though nutritionally poor, hay for horses, the lack of nutrients and energy being made up with a high-quality compounded nut or other concentrate. Where horses are affected they should be turned out to grass. If this is impracticable, hay should be soaked rather than merely dampened prior to feeding, or it should be replaced by silage, haylage or high-fibre compounded nuts. Straw bedding should be replaced by shredded paper or by softwood shavings and the ventilation of the box should be improved. Symptomatic horses may become asymptomatic in 4–24 days when horses are bedded on shredded paper and routinely fed a complete cubed diet. The pulmonary function values of asymptomatic horses may then not differ significantly from those of normal horses. The ingestion of mould spores, rather than their inhalation, as would occur with the feeding of soaked hay or nuts, is *not* a cause of the problem as it depends on a direct reaction between the inhaled particle and the lung alveoli. Meal feeding should also be avoided as some evidence indicates that allergic reactions may be seen to oat dust and some other sources of feed dust.

Crib-biting

Crib-biting is probably mediated by neurotransmitters and where there is a high frequency of the vice among horses it may indicate stress caused by bad management. A similar vice apparently does not occur among feral horses. Ways to palliate it include increased handling and exercise, a change of environment and feeding strategies.

STUDY QUESTIONS

- (1) How would you set about minimizing the risks of GI disturbances in a stable of working horses?
- (2) Where hyperlipaemia has been diagnosed in several pony mares, what is the appropriate course of action?
- (3) What are the causes of wasting disease in an elderly horse and what management should be instituted?

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Chapter 12

Laboratory Methods for Assessing Nutritional Status and Some Dietary Options

If the urine of a horse be somewhat high coloured, bright and cleare like lamber, and not like amber, or like a cup of strong March beere, then it sheweth the horse hath inflammation in his blood. . . . Now for the smell of his dung, you must understand, that the more provender you give, the greater will be the smell, and the lesse provender the lesse the smell.

G. Markham 1636

Many methods of nutritional assessment are only appropriate for large stables where normal variation in values can be overcome by determinations carried out in a number of horses fed in a similar manner. Nonetheless, whether one is dealing with individuals or groups, it is essential to establish normal values for the individuals or groups concerned. The normal value of a particular parameter in an individual may differ from the breed mean, as age, sex, time of year, system of management, stage of training, diet and breed all influence the norm. Thus, by establishing some routine practice it is possible to assess quickly whether a particular value has shifted from its norm, and this, in turn, enables one to uncover a disturbance in its early stages. Furthermore, observation and a range of tests are undoubtedly necessary truly to understand a situation.

Metabolic profiles have been adopted as measures of the nutritional and physiological health of dairy herds and some progress has been made towards similar techniques in stables. In assessing nutritional status, it is essential to have a full knowledge of the ingredient and chemical composition of all the feeds and the actual weights of each type fed over an extended period. Records and the retention of feed samples assist subsequent solution of problems. This procedure will facilitate an objective diet evaluation, which, with the monitoring of management and disease, should indicate the most appropriate laboratory measurements to be made. Direct and appropriate information should then come to hand and unnecessary expenditure will be avoided.

The adequate nutrition of the horse implies the normal nutrition of the tissues and cells of the body, but nutrient-related abnormality of these units does not necessarily imply incorrect feeding. A knowledge of the physiological and health status of the horse must be accommodated in any assessment. An improvement in health may not be achieved by dietary adjustment alone. It may require enteral or parenteral, nutrition to supplement or supplant normal diet for a limited period. A purpose of this chapter is to aid decisions in this direction.

METABOLIC TESTS

Many metabolic tests are now available for gauging nutritional status and some of the more commonly used measures are discussed in this chapter. It should, however, be emphasized that single measurements, let alone single determinations, are practically valueless. A salient reason for this is that few, if any, of these methods are specific in determining the status of a particular nutrient.

Variability of measured values

Repeat samples obtained from an individual horse normally yield variable values. This variability is caused by the inherent variability in any analytical method, in variable sample handling and variation within the horse while there may be no apparent variation in its health. Different analytical procedures for the same determinant yield different means, so it is important to quote the method employed with any set of values. To accommodate this variability, normal ranges for a particular parameter are quoted. Nonetheless, the important criterion is not the accepted normal range but the change in value from one day to another for an individual, as it could point to a trend in health. In order to detect such trends it is critical to control measurements so that real trends can be distinguished from cyclic variation.

Circadian variation in physiological characteristics has been well established as a phenomenon in man and many other animals. Several equine blood parameters show a biorhythmical pattern. Under natural conditions, horses feed throughout the 24-hour day, but tend to reduce their feeding activities at night, whereas under domestic management the principal meals are at set times. Feeding time is known to be a synchroniser of the glucose, NEFA and phosphorus rhythms. Some control of this effect can be achieved by, for example, sampling at the same time in the daily routine, preferably at a time when the rate of change is least and when environmental and metabolic factors of no concern have least influence. Meal-fed horses, like humans, show a fairly marked 24-hour circadian rhythm in the plasma concentration of PCV, plasma total protein, haemoglobin, urea, glucose, insulin, neutral fat, NEFA, cholesterol, Ca, P and several, frequently measured, enzymes (Greppi *et al.* 1996). For consistency of data, fixed daily times reduce variability in repeat samples for these characteristics, assuming the daily husbandry does not change.

Enzymes

Nomenclature

Over the years the names of many enzymes have changed, sometimes more than once. So that it is known which enzyme is referred to in any particular statement, the international Enzyme Commission has classified each identified and specific enzyme with its own code number, known, not surprisingly, as the EC number (International Union of Biochemistry and Molecular Biology on the Nomenclature and Classifica-

Table 12.1 Classification of enzymes.

Group	Name
1	Oxidoreductases
2	Transferases
3	Hydrolases
4	Lyases
5	Isomerases
6	Ligases

tion of Enzymes 1992). There are about 3500 enzymes on the list, which is still increasing in length. The EC number is a four-part system, the first number of which defines the class, the second the subclass of that class, the third the sub-subclass and the fourth the number it is given in the list of that sub-subclass, e.g. the oxidoreductase alanine dehydrogenase is defined as EC 1.4.1.1 and alanine aminotransferase as EC 2.6.1.2, as indicated in Table 12.1.

Specific tissue isoenzymes

The activity of many specific tissue enzymes is frequently measured as an aid to diagnosis. The plasma enzyme concentration may rise during leakage from the cells in which the enzyme is active, either as a result of maximum activity of those cells, e.g. muscle cells during galloping, or as a consequence of damage to the tissue, e.g. toxic damage of liver parenchymal cells. The location of the relevant tissue/organ is helped by measuring the isoenzyme concentration relative to a total raised activity. For example, there are at least five iso-forms of lactic dehydrogenase (EC 1.1.1.27). Isozymes are physically distinct, e.g. immunologically so, but they all catalyse the same reaction.

Diet and enzyme activity

Tissue enzymes are proteins that function only when present as the holoenzyme, which consists of the protein (apoenzyme that on its own has no catalytic activity) plus a heat-stable, dialysable non-protein coenzyme. This dialysable fraction contains nutrients in the form of certain water-soluble vitamers or vitamins A and K. Frequently, a metallic cofactor, e.g. zinc, is also needed. A dietary deficiency of one of these nutrients therefore depresses the activity of the specific enzyme(s) in which the nutrient functions.

The degree of saturation of several enzymes (the fraction present in the holoenzyme form), measured *in vitro* as the enzyme activity coefficient, can indicate the nutritional status in respect of the vitamin component of the cofactor (Table 12.2). It should be noted that the addition of the cofactor vitamer to the *in vitro* system will increase the activity regardless of the nutritional status, as a portion of the enzyme

Table 12.2 Cofactor vitamers of several tissue enzymes, with their EC numbers, of particular significance in equine nutrition and those* for which enzyme saturation measurements have been used to assess adequacy.

Enzyme	Cofactor vitamer	Nutrient in cofactor
Erythrocyte glutathione reductase, 1.6.4.2*	Flavin adenine dinucleotide	Riboflavin
Erythrocyte transketolase, 2.2.1.1*	Thiamin pyrophosphate	Thiamin
Pyruvate dehydrogenase, 1.2.4.1*	Cocarcboxylase (TPP)	Thiamin
Fatty-acid synthase ¹	Acetyl-CoA	Pantothenic acid
Glucose-6-phosphate dehydrogenase, 1.1.1.49	NADP(H)	Niacin
β -hydroxyacyl-CoA dehydrogenase, 1.1.1.35	NADP(H)	Niacin
Lactate dehydrogenase, 1.1.1.28	NADH and FAD	Niacin and riboflavin
Pyruvate carboxylase, 6.4.1.1	Biotin	Biotin
Erythrocyte alanine aminotransferase, 2.6.1.2*	Pyridoxal phosphate	Pyridoxine
Erythrocyte aspartate aminotransferase, 2.6.1.1*	Pyridoxal phosphate	Pyridoxine
Methionine synthetase, 4.2.99.10	5-methyltetrahydrofolic acid	Folic acid
Methylmalonyl-CoA mutase, 5.4.99.2	Adenosylcobalamin	Vitamin B ₁₂
α -carboxylase, 4.1.1.1	K hydroquinone	Vitamin K
Succinic dehydrogenase, 1.3.99.1	CoQ ₁₀	Ubiquinone ²

¹Multi-enzyme complex which differs among species.

²Not generally considered to be a vitamer because the molecular species shares an antioxidant role with other nutrients.

Also see vitamin status in 'Fat- and water-soluble vitamins', this chapter.

is normally present in the unsaturated apoenzyme form. Therefore the extent of the response is critical in assessing whether a dietary deficiency exists.

Serum enzymes and liver function

Clinical signs of hepatic insufficiency include loss of appetite, icterus, depression, weight loss, lethargy, yawning and, sometimes, wandering. Haemorrhages in oral mucous membranes have been reported and photosensitization may be observed, especially in horses with unpigmented skin and hair who are exposed to bright light. Photodynamic substances in the feed are incompletely metabolized by the compromised liver and cause hypersensitivity reactions in the skin.

Measurement of the serum activities of several hepatic enzymes is normally included in reaching a conclusion concerning the function and dysfunction of hepatic tissue. Increased serum activities of hepatic ALP, AST [formerly serum glutamic-oxaloacetic amino-transferase (SGOT)] and SDH (EC 1.1.1.14) may result from reversible changes in hepatocellular membranes, structural injury of hepatic tissue, caused by ischaemic necrosis or cholestasis, or from microsomal enzyme induction. Determinations of GGT (EC 2.3.2.2), serum bilirubin, serum proteins, the clearance of bromsulphthalein (BSP), excreted in the bile, and the histological examination of liver biopsy samples will also be appropriate in suspected cases of

hepatobiliary dysfunction. However, hepatobiliary disease is rare in horses, unless the common bile duct is obstructed.

Liver function is also assessed by plasma coagulation tests, including prothrombin time and partial thromboplastin time (platelet function tests, for which several instruments are available, should be evaluated in cases of bleeding problems). Increased liver-enzyme activity in serum can arise from decreased hepatic perfusion, causing hypoxia. Cardiac failure, endotoxaemia, septicaemia, hypothyroidism and hyperthermia may contribute to hypoxia of hepatic cells, but generally serum enzyme values do not rise more than two to three times the normal value, as a result of these extrahepatic causes.

Aspartate aminotransferase (AST, AAT, EC 2.6.1.1)

AST is a cytoplasmic and mitochondrial enzyme present in several tissues – liver and skeletal and cardiac muscles. The activity of this enzyme in serum/plasma is elevated most rapidly following acute hepatic necrosis and it may reach values 10–40 times normal, attaining a peak in 12–24 hours and declining over a two-week period. The enzyme may increase after larvicidal treatment with thiabendazole and in the hyperlipaemic syndrome of ponies and adult horses, but in chronic hepatic fibrosis the serum activity may be normal. The author (unpublished observations) has found values chronically 2–10 times normal in Miniature Shetland mares with bile-duct injury, but without hyperlipidaemia.

Alkaline phosphatase (ALP, EC 3.1.3.1)

(See also 'Bone metabolism', this chapter.) ALP is a membrane-bound enzyme synthesized in many tissues, notably active bone tissue, liver, kidney and intestinal mucosa (highest specific activity in the duodenum), although the intestinal source does not contribute significantly to total serum ALP in the horse. During ischaemic colic of the bowel, intestinal ALP is released into body fluids. A measure of this aids both diagnosis and a decision over immediate action in acute cases. Intestinal ALP is not bound by L-phenylalanine, whereas other sources are, thus providing a means of differentiation and detection of ischaemic colic when this specific isoenzyme is shown to be elevated in peritoneal fluid (Davies *et al.* 1984). Serum values are elevated during bone growth as a consequence of osteoblastic activity, and in horses with renal hyperparathyroidism or with NSHP in Ca or even in P deficiency. When hepatic injury accounts for the increase, it is frequently caused by cholestasis or primary bile-duct injury and the serum level often remains elevated with severe chronic disease.

Sorbitol dehydrogenase (SDH, EC 1.1.1.14)

SDH is concentrated in hepatocytes and so is used to demonstrate hepatic necrosis, but the serum activity declines in two to four hours following hepatic necrosis, and it has relatively low stability in stored samples. Nevertheless, elevated values indicate acute hepatic necrosis, and along with a raised AST they point to an hepatic origin for the disease.

Gamma-glutamyltransferase (GGT, EC 2.3.2.2)

GGT is membrane-bound in hepatic cells, but it also occurs in kidney and pancreatic tissue. However, elevated serum concentrations are accounted for almost entirely by release from bile-duct cells and serum increases result from intra- or extrahepatic cholestasis. Cholelithiasis is such a cause. The source of urinary GGT is probably the brush border of the proximal tubular renal epithelium. High urinary concentrations may indicate proximal tubular dysfunction. Rudolph & Corvalan (1992) found urinary GGT concentrations of 47.6 ± 27.3 iu/l in horses positive for proteinuria and 27.2 ± 17.2 iu/l in those negative (NB: some proteinuria is a physiological consequence of a high-protein diet).

Serum enzymes and exercise*Aminotransferase and creatine kinase (CK, EC 2.7.3.2)*

AST, alanine aminotransferase (ALT) (EC 2.6.1.2) and CK can be detected in the serum and tissues of the horse, although different isoenzymes of AST and ALT are found in the mitochondria and in the soluble portion of the cytoplasm of cells. In the absence of severe tissue injury, the cytoplasmic forms predominate in serum. These two enzymes require pyridoxyl 5'-phosphate as a cofactor for activity.

There is a large individual variation in the normal serum activity of these enzymes and individuals with apparently high circulating levels may achieve a good running performance. Nevertheless, where the intention is to determine whether the concentration within the peripheral circulation of, for example, a transferase, is within physiological limits, and not elevated to pathological concentrations, a wide margin of uncertainty must be allowed, unless there are good previous data available on a particular horse. It is also necessary to ensure that circulating apoenzyme is included in the total. For this, exogenous pyridoxal phosphate should be included in the assay medium (Rej *et al.* 1990). Serum concentrations increase slightly post exercise and are also elevated in disorders of muscle soreness, azoturia or 'tying-up' myositis.

Equine rhabdomyolysis is a muscle disorder resulting in elevated serum levels of muscle enzymes, stiffness, loss of performance and/or lameness, with a slightly higher frequency in two- and three-year-old fillies than in other horses. Serum concentrations of muscle enzymes 24 hours following exercise may more easily allow differentiation between muscle damage and the post-exercise physiological elevation. The 24-hour upper limits of normality may be taken as 100 and 300 iu/l, respectively for CK and AST. CK is relatively muscle specific and has a $t_{1/2}$ of about two hours, whereas AST has a $t_{1/2}$ of seven to eight days.

Plasma proteins

Albumin is the major plasma protein (Table 12.3) synthesized in the liver and it determines plasma colloidal osmotic pressure. Low serum values may indicate that over half the liver has been non-functional for several weeks, as albumin has a long half-life in plasma. Causes include trauma, malnutrition, parasitic-worm infestation,

Table 12.3 Blood serum albumin concentrations in TBs determined by the sulphate/sulphite method of Reinhold (1953).

	Number sampled	Range (g/l)	Mean (g/l)
Foal	140	12–45	25.8
Yearling	70	12–42	28.5
Two-year-old	120	9.5–42	26.8
Three-year-old	90	13.5–49	30.3
Four-year-old	52	18–47	29.8
Mare	32	18.5–52	29.6

protein-losing enteropathy and renal dysfunction, with elevated urinary levels, or poor hepatic circulation through hypotension, hypovolaemia or chronic inflammatory processes. Insufficient dietary protein is more likely to be the cause of depressed blood haemoglobin than is a relatively scarce quantity of dietary iron, where normal dietary ranges are concerned.

Plasma minerals

The plasma concentration of minerals is used, with considerable reservations, to assess the status of nutrients. These include Mg, K (but see p. 53, 62), P, Zn, Se, Cu, and T_3 and T_4 , reflecting I status. Cr is a component of the glucose tolerance factor and there is speculative evidence that depressed plasma Cr, or a measure of its renal excretion rate, may relate to glucose tolerance and insulin sensitivity. Serum ferritin provides a good index of hepatic and splenic Fe and can be used to evaluate Fe storage in horses. Whereas a deficiency of Se is characterized by a depression in serum concentration of the element and a depressed activity of Se-containing serum GSH-Px (EC 1.11.1.9), the situation with Zn is unclear. In growing children, dietary deprivation of Zn causes a depression in the serum concentration of the element only where there is sufficient dietary protein to promote a normal growth rate. In other species, at least, the activity of serum ALP (EC 3.1.3.1) is sensitive to Zn status, as Zn is a cofactor. The relationship in the horse has not been studied in detail, and the activity per se is not specific for Zn status (see page 71). Plasma concentrations of inorganic phosphate reflect the dietary intake of P and in the deficient state there is an *increase* in the activity of plasma ALP. Plasma measurements of inorganic phosphate and Ca are necessary, together with the urinary measurement of P, in the clearance test for Ca adequacy to be discussed in 'Urinary fractional electrolyte excretion (FE) test (creatinine clearance)', this chapter.

Intracellular minerals

When there is, for example, a dietary protein deficiency, some success has been achieved in detecting Zn inadequacy by measuring the more labile Zn content of

Table 12.4 Range in leucocyte Cu and Zn contents in seven Shetland ponies receiving poor hay only, before and after supplementation with Cu and Zn for 50 days (author's unpublished observations, by method of Williams *et al.* 1995).

	Cu ($\mu\text{g}/10^{-9}$ cells)	Zn ($\mu\text{g}/10^{-9}$ cells)
Before supplementation	0.11–0.18	2.57–6.25
After supplementation	0.40–2.86	2.57–10.57

leucocytes (Table 12.4). The reason for this is that Zn is a cofactor of cellular superoxide dismutase (SOD) (EC 1.15.1.1). There are two known forms of this enzyme: one contains Cu–Zn and is found in the cytoplasm of most cells and the other contains Mn and is present in the mitochondrial compartment of cells. The measurement of the leucocyte activity of SOD (EC 1.15.1.1), or the cellular content of Zn and Mn, which tends to reflect the activity of the enzyme, is a measure of the adequacy of these elements. For similar reasons the measurement of leucocyte or platelet Cu, required as a cofactor in cytochrome c oxidase (CCO, EC 1.9.3.1), or the cellular activity of CCO, is informative as a measure of Cu status. Plasma Cu is largely present in caeruloplasmin (EC 1.16.3.1), an acute-phase protein, the concentration of which relates to the inflammatory process, so plasma Cu concentration is unrelated to status.

Fat- and water-soluble vitamins

Among the fat-soluble vitamins the measurement of plasma α -tocopherol in relation to plasma lipid is a measure of storage status, and elevated breath alkanes may indicate deficiency. A deficiency of vitamin E and/or Se can cause muscle and liver damage that non-specifically leads to an increase in the activity of certain plasma enzymes, particularly AST and CK, owing to leakage from the tissue cells concerned. A more specific measure of adequacy is, however, the determination of red-cell fragility in the presence of dialuric acid or hydrogen peroxide (see Chapter 4 for a fuller discussion).

The homeostatic control of many blood components and the interaction of many nutrients imply that variations in blood concentrations require careful interpretation. Plasma retinol (vitamin A) concentration is only marginally informative. However, the concentration of vitamin A in the liver varies more closely with the dietary intake of vitamin A. Liver tissue is not easily accessed and an alternative sensitive measure of vitamin A adequacy is the RDR, determined in the horse's jugular blood and discussed in Chapter 4. Vitamin D deficiency occurs only in horses confined indoors for long periods, e.g. pit ponies, or those in the higher northern latitudes in winter. Status may be assessed by plasma concentration of 25-(OH)-D₃, possibly in conjunction with serum ALP (EC 3.1.3.1) activity. The determination of blood prothrombin time as a measure of vitamin K status is discussed in Chapter 11.

Elevated times would generally indicate antibiotic misuse, causing depressed intestinal synthesis of the vitamin.

Among the B vitamins, quick and routine methods are now available for assessing whole blood vitamin B₁₂ (cyanocobalamin), or erythrocyte B₁₂, and leucocyte folic acid, which, in the experience of the author, clearly reflect diet. Serum assays of folic acid frequently do not differentiate between the oxidised and reduced forms, so that a deficiency of the active, reduced, form may be masked. Piercy *et al.* (2002) overcame this by either measuring red-cell folate concentration, or by performing high performance liquid chromatography (HPLC) to determine the relative proportions of the active and inactive forms (see Chapter 4).

In the human subject the most sensitive indicators for detecting and separating B₁₂ and folate deficiencies are the measurements of methylmalonate and homocysteine concentrations in urine and blood. Elevation of both these metabolites indicates a B₁₂ deficiency, whereas an elevation in homocysteine only indicates a folate deficiency. Among other B vitamins, methods have been successfully developed for assessing thiamin, riboflavin and pyridoxine adequacy through measuring the activity coefficient of the appropriate enzymes discussed in 'Diet and enzyme activity', this chapter. The activity per se (not the coefficient) of a transaminase in red blood cells of sow pigs has been proposed as a good indicator of pyridoxine status. The *serum* activity of AST (AAT, EC 2.6.1.1), routinely measured in horses, is greatly influenced by the extent of leakage from liver and muscle cells and therefore the activity in horse red cells may reflect pyridoxine status. Biotin, a cofactor in carboxylases, is assessed in humans from whole blood or urinary biotin concentrations.

Tests for liver and kidney disease

Blood urea

Urea is derived from ammonia and, although the liver has a large over-capacity for its synthesis, blood urea values can be half normal with severe chronic liver disease. Blood values will also be greatly elevated when daily protein intakes exceed NRC requirements by a large margin, a not infrequent occurrence in TBs during training. Nevertheless, much ammonia produced during exercise has a different origin (see 'Ammonia and the alanine vehicle', Chapter 9).

Blood ammonia

Blood ammonia may derive from the action of bacterial urease on urea in the gut, although much of this ammonia will be reutilized in bacterial protein synthesis. Ammonia also results from the deamination of amino acids. Ammonia is converted to urea in the liver, but raised plasma levels have been noted in hepatic encephalopathy. The range of normal values is 80–160 µg/l. However, as blood samples must be placed on ice (not dry ice) immediately following collection and the plasma separated within 30 min, this measurement is problematic from a practical viewpoint.

Hepatic encephalopathy

Hepatic encephalopathy is a neurological dysfunction resulting from acute, or chronic, liver disease, characterized by depression, frequent yawning, muscle twitching, poor coordination, head pressing and loss of strength and posture. It is associated with high concentrations of blood ammonia, low blood glucose and increased levels of blood VFAs.

Bile acids

About 75% of bile acids in the horse are accounted for by chenodeoxycholic acid. Approximately 85% of these bile acids are conjugated to taurine and the remainder to glycine. The plasma content of total bile acids determined by HPLC rises slightly two to six hours after feeding, primarily owing to an increase in glycocholic acid production. Liver failure, obstruction of bile flow, or vascular shunting causes a rise in plasma bile acids, as a proportion of those absorbed from the gut is not removed by the liver for resecretion. Normal serum bile acid concentrations can be up to approximately 12 $\mu\text{mol/l}$, whereas in horses with clinical signs of liver disease the serum concentration may be more than double this level.

Bilirubin

Bilirubin is synthesized from the degradation of the haemoproteins of red blood cells in the reticuloendothelial cells of the spleen and liver. Bilirubin conjugated with glucuronic acid is excreted in the bile, except where hepatobiliary obstruction occurs and jaundice develops. Icterus is apparent when total plasma bilirubin concentration exceeds 34 $\mu\text{mol/l}$ (20 mg/l). ALP and GGT are also usually elevated. Fasting hyperbilirubinaemia can occur in the horse, possibly caused by a reduction in hepatic blood flow.

Reticuloendothelial-system (RES) function

The cells of the reticuloendothelial system (RES) have several functions. They act as a sieve, protecting the systemic blood circulation from some products carried to the liver by the portal system. These products include bacterial antigens of gut origin and both enterotoxins and endotoxins resulting from grain overload. It is thought that where there is a systemic acid-base imbalance and liver perfusion may be compromised, the sieve is then partly bypassed and clinical endotoxaemia may result.

Crystalline urine deposits

Urolithiasis is uncommon in the horse despite the supersaturation of equine urine with calcium carbonate. The most common site of calculus formation is the bladder and cystic and urethral calculi have been reported, whereas renal and ureteral

calculi are rare. Calcium carbonate is the predominant mineral of equine urinary calculi, although oxalate and phosphate types exist. Calculi in the horse may result from the mineralization of a nidus, possibly provided by a prior disease, such as pyelonephritis, renal papillary necrosis or tubular necrosis. In other species, urolithiasis typically develops with diets with low Ca:P ratios and excessively high, or low, Mg contents. Low Ca:P ratios activate calcitonin secretion, bone mobilization and soft-tissue calcification, leading to both nephrocalcinosis and urolithiasis. The situation in the horse differs in that the urine is normally supersaturated with Ca salts and persistent stone formers may lack some inhibitor of crystallization.

Urinary fractional electrolyte excretion (FE) test (creatinine clearance)

Electrolyte status is a function of intestinal absorption, renal tubule reabsorption, tissue deposition and mobilization, sweat loss and renal excretion. Serum or plasma concentrations of electrolytes may not be used to detect electrolyte imbalance, owing to efficient homeostatic mechanisms which maintain relatively normal blood concentrations despite extreme body depletion. Homeostasis is primarily mediated by the kidney, so that the amount of an electrolyte excreted daily varies with the body's status. However, the daily urine volume varies considerably between individuals so the concentration of an electrolyte in the urine is an unreliable guide to nutritional status. The collection of urinary losses over a period of several days would indicate the quantities surplus to requirement, but such extended urinary collection is impractical. Urine concentration of an electrolyte is therefore related to that of a control substance. This substance should (1) have an excretion rate similar to the glomerular filtration rate, and (2) not be secreted or reabsorbed by the renal tubules. Creatinine, the excretion product of creatine metabolism, fulfils these requirements reasonably well and the fractional electrolyte excretion (FE) is measured as the creatinine clearance ratio (renal creatinine averages 1.15 ± 0.41 mg/kg BW/hour; Meyer 1990). Urine sample creatinine concentrations of <9000 mmol/l may indicate contamination of the sample or excessive salt consumption, causing polydipsia/polyuria, so the FE values obtained should not then be accepted, although low values in horses of less than 18 months old may indicate a physiological abnormality (P.A. Harris 1996, personal communication).

A urine sample and a serum sample are required. The urine sampling should be achieved without resort to the use of diuretics because they affect Na and, to a lesser extent, K and chloride losses. The clearance of an electrolyte equals the concentration in the urine times the urine volume divided by the concentration in the serum. The clearance ratio is the clearance of the electrolyte divided by the clearance of creatinine. In this ratio, the urine volume cancels out and so need not be measured. The equation for which the determinants are required is given below:

$$\text{Percent electrolyte clearance (\% creatinine)} = \frac{[E]_u \times [\text{Creat}]_s}{[E]_s \times [\text{Creat}]_u} \times 100$$

where $[E]_u$ is the concentration of electrolyte in urine, $[E]_s$ is the concentration of electrolyte in serum, $[Creat]_s$ is the concentration of creatinine in serum and $[Creat]_u$ is the concentration of creatinine in urine.

High-concentrate rations tend to give a raised phosphate clearance and a depressed K clearance, the reverse being the case for high-roughage rations. These effects are quite normal. K clearance, together with measurements of blood and urinary pH, is useful in evaluating the type of acidosis and in assessing K depletion in exhausted horses, as the urinary excretion of K and H ions tends to display a reciprocal relationship. K clearance is depressed in chronic laminitis. Note that even slight blood haemolysis may increase plasma K and therefore yield falsely low FE values. A raised Na clearance may indicate excessive Na intake in the form of common salt, Addison's disease, dehydration or tubular malfunction.

Owing to the circadian rhythm of electrolyte excretion, referred to in 'Variability of measured values', this chapter, the FE ratio should ideally be measured at least three times during a 24-hour period. The serum and urine samples do not have to be taken simultaneously, but the samples should be taken pre-meal and pre-exercise. Decreased urinary Mg and Ca concentrations occur shortly before and after feeding with maximum values four to eight hours after feeding. The serum sample should be sterile and that of urine as close to sterility as possible. The urine sampling may benefit from some advice and it should comply with the following restrictions (P.A. Harris, Animal Health Trust, Newmarket, personal communication; Meyer 1990):

- Samples should be freely voided, using a collection harness before exercise (entire product of micturition). Walking the horse slowly first thing in the morning while its stable is being cleaned and rebedded may promote urination on return. If catheterization is necessary, fillies and mares should first be bled and then given a short brisk trot before sampling. Catheterization will decrease the risk of contamination, and so be helpful. Plasma K concentrations also decrease during eating and rise to a peak several hours post-ingestion.
- Meyer (1990) found that the mean urinary concentration of creatinine tended to be lower during the first two hours after eating. Thus, for the assessment of dietary adequacy by measurement of the ratio, the measurements should be made before feeding or at the following times after a meal:

3 hours	for Na
6–10 hours	for Cl
3–8 hours	for K
4–8 hours	for Ca
- There is little difference between the early, middle or late portions of a voided urine stream in the FE of Na, K, PO_4 and Cl, but for Ca collect the entire voided sample. Owing to the supersaturation of urinary Ca, reliable urine sampling for Ca clearance may not be achieved satisfactorily. The amount of Ca precipitate voided at any one moment is unpredictable. However, where a low urinary Ca value is supported by a low Mg value there is strong evidence of a dietary Ca

deficiency and possibly a deficiency of Mg. The reason for this is that Mg is more soluble so that urinary values are more reliable and Mg responds in a parallel fashion to that of Ca. The FE test may not be used to monitor low phosphate intakes.

- If a delay between collection and analysis is unavoidable, there is little change in the Na, K or Cl concentration in plasma, or urine, with storage at 18°C, 4°C or –20°C. However, considerable changes in Ca, PO₄ and creatinine concentrations occur, especially at 18°C. Hygiene is important. Samples should be transported in capped sterile containers and stored at 3–4°C for short periods, or at –20°C for longer periods. Ca, PO₄ and creatinine should be analysed as soon as possible, i.e. within four days.
- For urinary Ca determinations, mix well and use a flame atomic absorption spectrophotometer, as colorimetric methods used for serum or plasma samples are unsuitable.
- Samples collected during, or shortly after, an equine rhabdomyolysis episode do not reflect electrolyte status, owing to circulatory disturbances and to raised plasma myoglobin concentration which may affect renal function. Samples with a pH of 6 or below or those that are positive for glucose, blood, myoglobin and/or haemoglobin are unsuitable. A low urinary pH is accompanied by an increase in Ca and Mg excretion, giving falsely high Ca and Mg FE values. Blood and urinary pH may be decreased on a high-starch diet, yielding raised urinary Ca and P clearances, unrelated to changes in DCAB (see Chapter 9).

Effect of diet on FE

Diets with a Ca:P ratio of 1:1, or lower, produce an elevated FE PO₄, whereas inadequate Ca and low Na intakes lead to low FE Ca and low FE Na. Normal clearance ratio values are given in Table 12.5. Frequently TBs in hard training in the UK appear to be receiving inadequate Ca and Na, reflected in low FE values for Na and high values for PO₄. However, it is unclear whether there is any relationship between these inadequacies and the frequency of muscle stiffness, as the appropriate definitive experiments do not seem to have been conducted.

Table 12.5 Interquartile (25–75%) normal reference ranges for FE% (or creatinine clearance, % of electrolyte) of adult horses at rest and in work from spot samples (P. A. Harris personal communication)* and a mean published range.

Electrolyte	Grain based diet* FE %	Balanced compound and hay diet* FE %	Published FE %
PO ₄	0.0–0.5	0.0–0.2	0.04–1.19
Na	0.02–1	0.04–0.52	0.01–1
K	15–65	35–80	15–75
Cl	0.04–1.6	0.7–2.1	0.04–1.65
Ca		>7 (8–24)	
Mg		>15	

DIETS FOR LIVER DISEASE

Dietary causes of liver disease include aflatoxin, from mould-infected grains and protein concentrates (especially groundnuts), and pyrrolizidine alkaloid poisoning from *Senecio* and *Crotalaria* species. Supportive therapy has the objective of allowing time for regeneration of hepatocytes. This may initially involve i.v. glucose administration, followed by enteral feeding of glucose. For horses suffering severe hepatic dysfunction dietary management should ensure the following:

- The ration should be divided into at least three daily meals.
- The ration should contain the highest quality protein in adequate, but not excessive amounts.
- An amino acid supplement of the glucogenic branched chain amino acids isoleucine and valine (1 g/kg diet of each) may help.
- Soluble fibre sources, such as citrus pectin and beet pulp, are useful, together with wheat bran, soya hulls and other insoluble fibre sources, and a moderate level of several cooked starch sources.
- Fat supplements should not be added to the diet (although there may be a case for fats containing n-3 fatty acids, e.g. fish oils).
- Vitamin E (1500 iu/day) and a water-soluble B vitamin supplement, including 1000 mg choline/kg diet, are advisable.
- A supplement of 0.5 kg DL-methionine/tonne, has been recommended. There is, however, some evidence that excessive dietary methionine is converted by gut bacteria to mercaptans. Following absorption a diseased liver is unable to clear these bacterial metabolites adequately and, acting with ammonia, they may cause signs of encephalopathy. Unfortunately mercaptans are also derived from cystine, so, if the evidence is reliable, the basal diet should be relatively low in total sulphur amino acids, i.e. not more than 3.5 g/kg diet. If methionine is to be added it should ideally be the L- α -isomer (preferable to DL-), and included at a concentration of 0.25 kg/tonne. (The author has never observed encephalopathy to result from methionine supplements in horses with compromised livers and so the risk may be slight.)

DIETS FOR KIDNEY DISEASE

Renal dysfunction and failure can be caused by any acute condition leading to a severe reduction in glomerular filtration rate. This can be induced by shock, impairing renal blood flow, trauma and haemorrhage, severe dehydration, obstruction of the urethra, hypersensitivity reactions, certain bacterial and mould toxins and consumption of particular metal salts, including those of mercury, lead, arsenic and hexavalent chromium (see also Chapter 3).

Renal failure may lead to metabolic acidosis, therefore the diet should have as an objective the prevention of a low urinary pH. Dietary protein should be as described for liver disease (see 'Diets for liver disease' above), with an emphasis on protein of excellent amino-acid balance, given in adequate, but not excessive, quantities. Thus,

deamination and uraemia will be minimized. A cooked, high-carbohydrate diet should be given to provide an adequate energy supply, so avoiding endogenous protein catabolism. Dietary P should not be excessive and the Ca:P ratio should be 2:1. Sodium chloride supplements should not be given in excessive amounts and diuretics should be avoided. Potassium retention can occur, so that control should be exercised over dietary potassium. Grasses and other raw materials known to contain oxalates should be excluded, and a pyridoxine supplement might be given. Fresh water should be available at all times. Blood urea and ammonia should be monitored in both kidney and liver dysfunction.

BONE METABOLISM

Skeletal ailments are not infrequent in young growing stock and chronic, non-specific lameness in adults is sometimes an expression of NSHP, by far the most common cause of which is faulty Ca and P nutrition and an improper balance between these two minerals. A simple means of evaluating Ca adequacy is therefore required. Ca concentrations in serum vary to a small degree in relation to Ca intake, and where they are measured in a number of horses, significant differences can be detected between deficient and normal groups (Fig. 3.2, p. 53). However, the method is insufficiently sensitive to be of practical use. Greater sensitivity is achieved with creatinine clearance ratios. The horse seems to regulate serum Ca more by renal excretion than by controlling intestinal absorption. Intact proximal tubules are required for P reabsorption and Ca excretion and in renal failure serum Ca is increased and P is depressed. The fully functioning kidney, on the other hand, excretes excess Ca in the urine and therefore an inadequate intake might be revealed by a reduced urinary excretion, if it were not for the fact that Ca sediments in horse urine, owing to its alkaline nature. Repeatable estimates of the Ca content of urine cannot therefore be readily attained.

Phosphate clearance may fall to zero where the diet is only marginally adequate in P and the clearance will be increased when the intake is greatly in excess of need, or when the horse is suffering from NSHP. An inadequate dietary intake of Ca stimulates the secretion of parathyroid hormone (PTH), which increases the reabsorption of Ca by the renal tubules, mobilizes bone Ca, and increases the loss of phosphate through the tubules by decreasing tubular reabsorption of phosphate (see 'Vitamin D' Chapter 4). The net effect of this is to stabilize the ionized concentration of serum Ca and to depress serum phosphate. Concentrations of Ca in serum tend to be below average, but still within the normal range. An increased phosphate clearance with a normal or slightly depressed serum Ca, is therefore frequently indicative of insufficient dietary Ca when other causes have been eliminated from consideration. The method, nevertheless, has its limits.

Measurement of changes in the rate of bone metabolism would be helpful in assessing bone modelling and remodelling, during training, in the detection of cases of OCD or early stages of other bone disorders and during fracture repair. Bone ALP is an enzyme located on the cell surface of osteoblasts and it plays an important

Table 12.6 Reference ranges in serum from healthy horses for PICP, ICTP, bone ALP and total ALP (after Price *et al.* 1995).

Age group	PICP ($\mu\text{g/l}$)	ICTP ($\mu\text{g/l}$)	Bone ALP (u/l)	Total ALP (u/l)
<1 year	1216–2666	14–27	134–288	223–498
1–2 years	550–1472	8–23	33–125	134–238
3–4 years	248–925	6–15	25–70	101–203
5–20 years	136–394	0–9	13–47	91–352

role in bone formation. It accounts for approximately 60% of circulating ALP activity in horses of less than one year of age, whereas in those over five years it accounts for about 20%. Separation of the bone isoform is therefore necessary in the detection of changes in bone metabolism.

Price *et al.* (1995) reported procedures for the measurement of bone ALP in serum by precipitating it with wheat-germ lectin (NB: liver ALP binds with wheat germ in man, but not in the horse, and two-thirds of caecum ALP binds in the horse, but does not appear in the blood). This binding depends on the number of carbohydrate units in the side chain of the enzyme, and, although there are two or three isotypes within each tissue of origin, the procedure still segregates tissue sources. Using radioimmunoassay, Price *et al.* (1995) also measured serum carboxyterminal propeptide of type I collagen (PICP) and serum pyridinoline cross-linked telopeptide domains of type I collagen (ICTP). ICTP is liberated into serum during the collagen degradation of bone resorption in man and it is a precursor of pyridinoline cross-links in urine. These determinants are all associated with bone modelling and are quantitatively and inversely correlated with age, at least up to five years in the horse (Table 12.6). Deviations from these ranges probably indicate bone metabolism, related to bone disease or repair, and with the parallel determination of other parameters, particularly Ca and P clearance, the data should be of considerable diagnostic value. Type I collagen also occurs in the skin, tendons and ligaments, but the proportion in the serum pool, derived from these sources, seems to be small, and moreover inflammation of tendons in the horse is frequently related to bone resorption.

Osteocalcin has been used to measure bone metabolism, but the molecule is extremely labile and assay values in different laboratories have varied considerably (see 'Later growth and conformational changes', Chapter 8).

OTHER TESTS

Hair analysis

The chemical analysis of hair samples has been suggested as a means of measuring intakes of proteins, minerals, trace elements and toxic heavy metals. The author has

found increased amounts of lead in hair in lead toxicosis. However, the mineral composition is influenced by hair colour (Hintz 1980b) and the intake of both minerals and trace elements may be more reliably quantified by other means. The bulb diameter of hair among horses under a range of conditions has been used successfully in the assessment of dietary protein adequacy (Godbee *et al.* 1979).

Sugar tolerance (as distinct from the glucose tolerance test, GTT)

Glucose and xylose absorption tests have been used to measure the gross function of the small intestine. The recommended procedure is to give 0.5 g D(+)xylose/kg BW as a 10% solution by stomach tube. By measuring the peak plasma xylose concentration after 90 min it is possible to discriminate between normal and abnormal absorption. A normal control animal should also be measured under the same conditions. An oral lactose tolerance test (1 g/kg BW, as a 20% solution) may help determine small-intestinal mucosal damage in diarrhoeic foals, when the continued ingestion of lactose may be detrimental (Roberts 1975a,b; see also Chapter 1).

Feed allergen tests

Feed allergen tests have been used in horses successfully by the author for determining whether feed entities are causal in certain oedematous skin and respiratory irregularities. The horse seems to be prone to such reactions, but cross reactions between feeds must be expected. Blood and skin tests are appropriate in the detection of dietary and mould allergens, although the intradermal skin test has shown poor repeatability (Lebis *et al.* 2002). Nevertheless, the method detected a positive response to *Culex pipiens* (common grey gnat, a mosquito) and to *Dermatophagoides farinae* (sarcoptiform mite) in horses with skin signs of supposedly allergic origin.

Haematology

The measurement of numbers of red and white blood cells has been used in folic acid, vitamin B₁₂ and protein assessments. For consistency, samples should be drawn immediately after strenuous exercise; lower erythrocyte parameters should be expected for ponies and cold-blooded horses. Normal values are also influenced by sex, age, stable and season, and, among racehorses, by training level. More sensitive methods of assessing vitamin B₁₂ and folic acid status are suggested in 'Fat- and water-soluble vitamins', this chapter.

Potassium status

(See also Chapter 11.) The measurement of [K⁺] in red blood cells should be undertaken within two hours of removal of the blood from the horse. The metabolic energy required for the active transport of K⁺ is not generated in blood samples

Table 12.7 A sequence of procedures for investigating possible causes of signs indicating possible nutritional anomalies in horses, recorded descriptively and by photograph, with times and dates.

Sequence of initial actions	Records to be kept	Laboratory investigation sequence
(1) Examination of horses, social interaction, collection of tissue samples, immediate therapy	Abnormal signs and age distribution	Routine haematology and biochemistry, preserve samples for later examination
(2) Examine co-grazing ruminants	Associated signs	If necessary, routine blood values
(3) Examine stable/paddocks	Water, weeds, pollutants, bedding, poisons, surface materials, dust	Identification of plants, pollutants and poisonous substances
(4) Sample all feeds, including pasture	Physical examination, ingredient make-up, calculate nutrient levels and intakes	Feed analysis for heavy metals, drugs, trace elements, fats, natural toxins
(5) Collect previous feed samples and records	Calculate presumed nutrient intakes	Calculate chronic intakes of nutrients and toxicants Specific non-routine determinations on initial samples
(6) Test presumptive corrective treatments in sequence or in selected sub-groups of horses	Clinical results	Determine any change in fresh sample values, previously abnormal

stored in the refrigerator and consequently K^+ leaks by diffusion from the cells and Na^+ penetrates their membranes. This movement can be reversed by incubating the blood with glucose, or, in fact, by reinjecting the cells into the circulation.

PROCEDURES FOR DETERMINING CAUSES OF SUSPECTED NUTRITIONAL AND DIETARY PROBLEMS

Procedures for determining causes of suspected nutritional and dietary problems must vary according to the amount and reliability of the information at hand and whether there is a justification for determining causes rather than simply replacing an inadequate feed system by a proven system. A proposed procedure is given in Table 12.7.

STUDY QUESTIONS

- (1) Construct a 'decision tree' and procedure you would adopt for determining the causes of a diet-induced nutritional problem.
- (2) How would you propose a horse with poor liver function be fed and managed?

FURTHER READING

- Greppi, G.F., Casini, L., Gatta, D., Orlandi, M. & Pasquini, M. (1996) Daily fluctuations of haematology and blood biochemistry in horses fed varying levels of protein. *Equine Veterinary Journal*, **28**, 350–53.
- Price, J.S., Jackson, B., Eastell, R., *et al.* (1995) Age related changes in biochemical markers of bone metabolism in horses. *Equine Veterinary Journal*, **27**, 201–207.

Appendix A

Example Calculation of Dietary Composition Required for a 400 kg Mare in the Fourth Month of Lactation

(1) To calculate the proportions of hay and concentrate in the total diet, divide the required daily energy intake (MJ/day) by the desirable daily total feed intake (88% DM) to give the average energy density of the total diet. Then form a simple equation containing the energy densities of the roughage and of the cereal available (88% DM).

Required daily energy	84.5 MJ DE/day
Desirable total feed intake	9.0 kg
Oats	12.1 MJ DE/kg
Hay	7.3 MJ DE/kg

Let x be the proportion of cereal in the diet and $1 - x$ be the proportion of hay.

$$\begin{aligned}
 12.1x + 7.3(1 - x) &= 84.5 / 9.0 \\
 12.1x + 7.3 - 7.3x &= 9.39 \text{ MJ DE / kg} \\
 4.8x &= (9.39 - 7.3) \text{ MJ DE / kg} \\
 x &= 2.09 / 4.8 = 0.435 \text{ or } 43.5\% \text{ oats (435 g/kg)}
 \end{aligned}$$

If 43.5% of the diet is oats then 56.5% is hay.

(2) Now calculate the protein, Ca and P contents of this simple mix from the information given in Appendix C.

Oats (12.1 MJ DE) contains (per kg):

95 g	crude protein
0.8 g	Ca
3.3 g	P

Hay (7.3 MJ DE) contains (per kg):

55 g	crude protein
3.5 g	Ca
1.7 g	P

Initial dietary composition (g)

	Crude protein	Ca	P
Contributed by oats:			
$\frac{43.5}{100} \times \text{each oat value above}$	41.3	0.35	1.44
Contributed by hay:			
$\frac{56.5}{100} \times \text{each hay value above}$	31.1	1.98	0.96
Total initial composition (g/kg)	72.4	2.33	2.40
Requirement (g/kg)	120.0	5.00	3.00
Deficit of minerals (g/kg)	—	2.67	0.60

Now calculate how much soya-bean meal is required to make good the protein deficit (other protein concentrates could be used in amounts that are inversely proportional to the lysine contents of the protein source under consideration and that of soya). About 2–3 g more protein than is required should be allowed for because minerals will also be added to the diet. Soya-bean meal from Appendix C contains 440 g crude protein/kg. As before:

$$72.4x + 440(1 - x) = 120.0 + 3 \text{ g crude protein/kg}$$

$$72.4x + 440 - 440x = 123 \text{ g/kg}$$

$$-367.6x = -317 \text{ g/kg}$$

Change signs on both sides.

$x = 0.862$ or 86.2% oats plus hay, and therefore $(100 - 86.2)$ soya forms the remainder, i.e. 13.8% soya-bean meal.

The soya contains more energy than oats and hay, but this will be approximately compensated for by the complete absence of energy in the mineral and vitamin supplement.

(3) Now 0.6 g P/kg is still required, being the deficit shown above. Dicalcium phosphate contains 188 g P/1000 g (from Appendix C). Therefore an addition of:

$$\frac{1000}{188} \times 0.6 \text{ g} = 3.19 \text{ g dicalcium phosphate (CaHPO}_4\text{)/kg}$$

total feed will provide the necessary P.

Dicalcium phosphate from Appendix C also contains 237 g Ca/1000 g. Therefore 3.19 g provides:

$$\frac{237}{1000} \times 3.19 \text{ g} = 0.76 \text{ g Ca}$$

The original deficit was 2.67 g Ca and it is now:

$$2.67 - 0.76 \text{ g} = 1.91 \text{ g Ca}$$

This can be made up with limestone flour (CaCO_3) containing 360 g Ca/1000 g. Therefore:

$$\frac{1000}{360} \times 1.91 = 5.31 \text{ g CaCO}_3/\text{kg total diet}$$

(4) The diet should also contain 5 g common salt (NaCl)/kg and a proprietary vitamin/trace-element mixture suitable for horses.

(5) The complete diet is now as follows:

	g/kg	%
Oats	283	28.35 (43.5 – 13.8 – 0.32 – 0.53 – 0.5)
Soya-bean meal	138	13.80
Dicalcium phosphate	3.2	0.32
Calcium carbonate	5.3	0.53
Salt	5.0	0.50
Vitamins/trace elements	+	+
Hay	565	56.50
Total		100

* A supplement may be provided as described in Chapters 6 and 8.

The composition of the concentrate portion of the ration is as follows:

		Per cent
Oats	$\frac{28.3}{43.5} \times 100$	65.1
Soya-bean meal	$\frac{13.8}{43.5} \times 100$	31.7
Dicalcium phosphate	$\frac{0.32}{43.5} \times 100$	0.74
Calcium carbonate	$\frac{0.53}{43.5} \times 100$	1.22
Salt	$\frac{0.5}{43.5} \times 100$	1.15
Vitamins/trace elements		0.1
Total		100

(6) The total daily ration is to be 9 kg and of this the above concentrate would form $43.5/100 \times 9 = 3.9$ kg daily. The remaining 5.1 kg hay could be given in excess, as some

will be lost and horses would naturally consume their ration of concentrate before filling up on hay.

The concentrate ration should be divided into a minimum of two feeds per day and introduced gradually in increasing amounts until the full ration is provided. Small adjustments of the quantity can be made to allow for differences in condition between individuals. Where growing horses are being fed, particularly of faster growing breeds, the overriding concern must be the condition of the legs, and if there is any tendency towards contracted flexor tendons, epiphysitis or crooked legs the concentrate allowance should be reduced until the condition subsides (see Chapter 8 for details).

Appendix B

Common Dietary Errors in Studs and Racing Stables

The table below shows the range of dietary composition for foals and yearlings in ten studs where home mixes have been prepared and when restricted access to pasture of moderate quality is provided (% of total diet, air-dry basis; dashes imply none used).

	Dietary range		Typical poor-quality diets at specific studs			
	Weaned foals	Yearlings	Foals (1)	Foals (2)	Yearlings (1)	Yearlings (2)
Oats	37–70	11–58	48	—	57	9.5
Boiled barley	0–1.6	0–26	1.6	—	—	20
Flaked maize	0	0–6	—	—	—	2.5
Bran (wheat)	0–12	5–28	5	—	28	16
Coarse mix (sweetfeed)						
of low quality	0–43	0–43	—	42	—	—
Barley chaff	0–7	0–7	—	7	—	—
Cubes of moderate quality	0–12	0–60	12	—	—	23
Soya-bean meal	0–4	0	—	—	—	—
Linseed (boiled)	0–7	0–9	1.6	—	5	7
Palm-kernel cake	0	0–3	—	—	—	—
Milk pellets	0–13	0	—	—	—	—
Locust beans	0	0–3	—	—	—	—
Egg	0	0+	—	—	—	—
Molasses	0	0–2	—	—	—	—
Carrots	0	0–3	—	—	—	—

	Dietary range		Typical poor-quality diets at specific studs			
	Weaned foals	Yearlings	Foals (1)	Foals (2)	Yearlings (1)	Yearlings (2)
Grass hay	6–18	14–29	16	18	10	11
Lucerne hay	0–9.6	0	—	9.5	—	—
Limestone flour	0–2.2	0–2.2	—	2.2	—	—
Dicalcium phosphate	0–0.5	0	—	—	—	—
Vitamins, trace elements and minerals	+*	0–+	—	—	—	—
Pasture	12–36	14–40	16	22	—	11
* Some sort of supplement was used in each of the ten stables.						
<i>Some chemical characteristics</i>						
Crude protein (%)	10–16	10–17	11.1	10.0	11.8	11.7
Total lysine (%)	0.4–0.6	0.4–0.7	0.48	0.40	0.45	0.45
Ca (%)	0.32–0.9	0.22–1.2	0.33	0.65	0.23	0.44
P (%)	0.2–0.46	0.2–0.65	0.42	0.19	0.65	0.57

General comments:

- widespread use of poor-quality hay for young stock;
- failure to rectify this by adjustment of concentrate composition;
- insufficient checks on rates of growth;
- failure to compensate for inadequacies of pastures.

Common errors:

- very variable protein and lysine intakes exacerbated by variable pasture quality and availability;
- sub-optimum protein:energy ratios for weaned foals;
- extreme variation in Ca intake;
- sub-optimum Ca:P ratios;
- excessive intakes of vitamins A and D, but possible inadequacies of several other vitamins;
- lack of control of growth curve leading to poor conformation, epiphysitis and abnormal alignment of legs;
- use of poorly formulated micronutrient supplements;
- use of more than one micronutrient supplement lacking complementary effects;

- pasture trace-element and other deficiencies, which contribute to metabolic and conformational problems.

Faults in diets (1) and (2) specified above

Foals:

- (1) Insufficient protein, lysine and Ca.
Excessive use of vitamins A and D₃.
Deficiency of selenium and marginal zinc status.
- (2) Insufficient protein and lysine.
Excessively wide Ca:P ratio.
Selenium depletion, zinc inadequacy and suspected induced manganese deficiency.
Poor conformation.

Yearlings:

- (1) Marginal protein and lysine inadequacy.
Ca deficiency with adverse Ca:P ratio.
Epiphysitis evident.
Several vitamin inadequacies and suspect trace-element status.
- (2) Lack of objectivity in ration formulation and unnecessary complexity.
Adverse Ca:P ratio.

Range of dietary composition for TBs in eight racing stables where home mixes are prepared (% of total diet, air-dry basis; dashes imply none used, or analytical value unavailable).

	Dietary range	Typical poor-quality diets at specific stables	
		(1)	(2)
Oats	32–59	49	51
Bran (wheat)	0–16	—	1.1
Coarse mix (sweetfeed)	0–20	—	—
Chaff	0–3.7	—	—
Cubes	0–24	—	2.0
Soya-bean meal	0–2.2	0.8	0.7
Linseed (boiled)	0–1.5	0.8	1.5
Molassed peat	0–0.5	—	0.4
Carrots	0–2	—	—
Grass pellets	0–6	—	—
Molasses	0–1.5	—	1.4
Grass hay	28–53	49	40.6
Limestone flour	0–1.1	—	1.1
Salt (sodium chloride)	0–0.1	—	0.07

	Dietary range	Typical poor-quality diets at specific stables	
		(1)	(2)
Corn oil	0–0.57	0.11	—
Vitamins, trace elements and minerals	0–4	0.2	0.1
<i>Some chemical characteristics</i>			
Crude protein (%)	7.2–11.5	7.3	7.4
Total lysine (%)	0.35–0.5	0.36	0.40
Ca (%)	0.15–1.38	0.15	0.68
P (%)	0.24–0.43	0.25	0.28
K (%)	—	1.5	—
Na (%)	—	0.16	—
Mg (%)	—	0.18	—
Zn (mg/kg)	—	—	24
Mn (mg/kg)	—	—	46

General comments:

- oats and poor-quality grass hay frequently constitute over 90% of the diet and their composition is variable and unknown;
- several non-complementary micronutrient supplements are frequently used in the same diet;
- frequently insufficient common salt is consumed in hot weather;
- frequency of feeding is often insufficient;
- rate of ration change and of energy intake frequently inappropriate;
- notion that a rest on Sundays with changes in management and in feeding benefits the horse is misguided, in contrast to human benefits.

Common errors:

- variable protein and lysine intakes;
- sub-optimum protein:energy ratios;
- extreme variability in Ca intake;
- sub-optimum Ca:P ratios;
- excesses of vitamins A and D₃;
- inadequate allowances of folic acid and possibly of other water-soluble vitamins and of salt;
- incorrect quantities of vitamins, trace elements and minerals provided by most supplements.

Faults in diets (1) and (2) specified above:

- (1) Lameness, metabolic upsets (e.g. azoturia, set-fast).
Dietary faults include excesses of vitamins A and D₃ and excess iodine.
Insufficient Ca and adverse Ca:P ratio.

Insufficient sodium and folic acid, marginal protein and probably too wide an energy:protein ratio.

(2) Abnormal blood characteristics.

Marginally low protein intake.

Insufficient allowances of zinc, folic acid and salt.

Appendix C

Chemical Composition of Feedstuffs Used for Horses

Values for feedstuffs (a) assume 880g DM/kg (dashes imply no value available); values for forages (b) are typical rather than average values. Equations in (c), (d) and (e) can be used to estimate UFC values from the chemical composition of feeds.

(a) Chemical composition of feed stuffs (g/kg), assuming 880g DM/kg.

	Crude protein (g/kg)	MADC		Oil (g/kg)	Crude fibre (g/kg)	MAD fibre (g/kg)	NDF (g/kg)	Ash (g/kg)	Ca (g/kg)	P (g/kg)
		(g/kg DM)	(88% DM)							
Oats	96	103	91	45	100	170	240	40	0.7	3.0
Barley	95	92	81	18	50	70	167	25	0.6	3.3
Wheat	100	98	86	15	22	40	123	19	0.4	3.2
Maize	85	79	70	38	25	30	95	15	0.2	3.0
Sorghum (white)	10.6			25	27	60	201	18	0.3	2.7
Rice (rough)	73			17	90	—	—	52	0.4	2.6
Millet	88			13	277	—	—	81	—	—
White fishmeal	660			80	0	0	0	215	57.0	34.0
Dried skimmed milk (spray)	340			6	0	0	0	80	10.5	9.8
Linseeds	219			316	76	135	—	45	2.4	5.2
Exp. linseed meal	320	323	284	60	100	170	220	60	3.0	7.3
Extr. soya-bean meal	440	437	385	10	62	100	132	60	2.4	6.3
Extr. sunflower seedmeal	280–450	344	303	18	230	—	360–450	77	2.9	8.0
Exp. cottonseed cake	410			37	140	220	250	65	2.0	10.5
Groundnut meal extr. 48–50% CP	470	—	—	13	140	220	—	50	2.0	7.1
Maize gluten feed	210	191	168	25	70–80	—	—	70	2.5	7.5
Maize gluten feed (40%)	410	427	376	25	50	—	—	40	2.0	5.1
Field beans	255	272	239	90	74	114	—	29	0.8	4.8
Peas	229	223	196	50	57	82	172	27	0.7	4.0
High-protein grassmeal	160			32	220	360	540	70	6.0	2.3
Lucerne meal	170	101–122	89–107	30	170–250	400	410	100	15.0	2.0
Brewer's yeast	450			10	30	—	—	65	4.4	13.3
Rapeseed meal extr.	350			24	130	—	210	70	6.5	11.0
Wheatfeed	155	148	130	35	85	100	310	50	1.0	10.0
Wheat bran	155	130	114	30	110	120	380	70	1.0	12.1
Extr. rice bran	135			15	120	190	400	125	1.1	19.0

K (g/kg)	Lysine (g/kg)	DE (MJ/kg)	UFC		α -Toco- pherol (mg/kg)	Free folic acid (mg/kg)	Available biotin (μ g/kg)	Thiamin (mg/kg)	Riboflavin (mg/kg)	Pantothenic acid (mg/kg)
			(g/kg DM)	(88% DM)						
5.0	3.2	10.9–12.1	0.99	0.87	9	0.12	50	17.0	1.7	12.0
5.0	3.1	12.8	1.16	1.02	7	0.11	12	5.0	1.8	14.0
4.2	2.8	14.1	1.26	1.11	9	0.12	4	4.0	1.1	11.0
3.1	2.6	14.2	1.33	1.17	9	0.06	65	2.0	1.5	6.0
4.1	2.4	13.0	—	—	7	0.13	—	3.0	1.1	12.0
—	2.5	11.1	—	—	—	0.20	15	2.5	0.9	—
—	—	—	—	—	—	—	—	—	—	—
8.3	48.0	14.1	—	—	13	0.22	100	5.0	5.0	8.8
16.0	29.0	15.1	—	—	10	0.60	330	4.0	20.0	30.0
9.4	7.7	18.5	—	—	2	—	—	7.0	2.5	—
11.0	11.3	13.9	0.92	0.81	1	—	—	8.0	3.0	12.0
23.5	26.0	13.3	1.06	0.99	2	0.57	280	6.0	3.3	14.0
14.0	13.0	9.5	0.79	0.70	10	—	415	34.0	3.0	—
15.0	14.0	12.8	—	—	5	0.22	250	7.0	4.0	9.5
12.0	15.0	12.5	1.01	0.89	2	0.5	300	8.0	3.0	40.0
9.7	5.8	12.8	0.96	0.85	7	0.20	85	2.0	2.2	14.0
1.0	11.2	13.1	1.14	1	7	0.2	90	0.4	1.8	12.0
11.0	17.0	13.1	1.04	0.92	—	—	—	—	—	—
11.0	15.8	14.1	1.15	1.01	8	—	—	—	1.4	—
21.0	8.0	9.6	—	—	25	1.80	300	3.0	15.0	25.0
22.0	8.2	9.0–10.0	0.57–0.68	0.5–0.6	30	3.00	400	3.7	14.0	25.0
18.0	29.3	12.2	—	—	2	2.31	300	100.0	39.0	105.0
—	20.0	11.5	—	—	—	—	575	1.0	3.6	9.0
12.0	6.1	11.0	1.09	0.96	23	0.67	15	13.0	4.5	16.0
14.0	6.0	10.8	0.86	0.75	21	0.30	15	7.3	5.4	26.0
19.0	5.3	10.8	—	—	—	—	20	23.0	2.6	23.0

	Crude protein (g/kg)	MADC		Oil (g/kg)	Crude fibre (g/kg)	MAD fibre (g/kg)	NDF (g/kg)	Ash (g/kg)	Ca (g/kg)	P (g/kg)
		(g/kg DM)	(88% DM)							
Oatfeed	50			22	250	400	635	70	0.8	1.2
Malt sprouts (culms)	250	257	226	20	140	—	390	65	2.0	7.0
Dried brewer's grains	180–250	251	221	62	140–170	180	405	38	2.6	5.1
Soya hulls	114	—	—	20	370	440	586	43	5	1.7
Beet pulp	100	45	40	10	174	340	392	50	7.0	0.8
Molassed beet pulp	90–120			1–6	130	—	300	60–80	6.0	0.7
Grass hay	42–80	40–60	35–53	25	330	380	670	50	2.9	1.7
Matua grass hay	90–170	—	—	40–80	—	270–350	520–700	60–140	—	—
Grass haylage	62	24	21	—	—	—	—	—	5.1	1.8
Big bale silage	98–100	52	46	35	232	273	—	62	4.1	3.3
Clamp silage	108–135	73	64	28	299	334	—	62	4.8– 5.3	2.6– 2.9
Maize silage	70	—	—	—	—	245	430	45	2.7	2.0
Clover/grass hay	60–100	50–70	44–62	27	330	380	600	65	4.0	1.7
Maize silage in cob	70	30	26	—	—	245	430–760	45	2.7	2.0
Spring cereal straw, barley	30	0	0	19	410	590	640	70	2.0	0.4
Vegetable oil	0	—	—	100	0	0	0	0	0	0
Molasses (cane)	30	34	30	0	0	0	0	85	7.2	1.0
Molasses (sugar beet)	—	83	73	—	—	—	0	—	—	—
Nutritionally improved straw*	45	—	—	12	340	490	—	—	4.0	1.0
Limestone flour	0	0	0	0	0	0	0	990	365	4
Dicalcium phosphate	0	0	0	0	0	0	0	100	238	187
Steamed bone flour	0	—	—	0	0	0	0	980	323	133

K (g/kg)	Lysine (g/kg)	DE (MJ/kg)	UFC		α -Toco- pherol (mg/kg)	Free folic acid (mg/kg)	Available biotin (μ g/kg)	Thiamin (mg/kg)	Riboflavin (mg/kg)	Pantothenic acid (mg/kg)
			(g/kg DM)	(88% DM)						
7.0	1.9	7.7			—	—	—	—	—	—
—	12.0	10.0	0.92	0.81	—	—	—	—	—	—
1.0	8.3	10.0	0.79	0.7	10	—	—	0.6	1.0	10.0
10	4.6	6.9	—	—	—	0.5	—	1.5	3.5	13
2.0	5.6	11.0	0.86	0.75	—	—	—	0.4	0.6	1.5
16.0	2.6	12.0	—	—	1	—	—	0.4	0.4	2
17.0	3.0	5.0–7.4	0.41–0.55	0.36–0.48	7	—	—	1.5	10	—
—	—	—	—	—	—	—	—	—	—	—
—	—	8.0	—	—	—	—	—	—	—	—
—	—	8.7	0.65–0.72	0.57–0.63	—	—	—	—	—	—
15	—	9.2– 10.5	0.60–0.68	0.53–0.60	—	—	—	—	—	—
—	3.8	4.49	—	—	—	—	—	—	—	—
19.0	5.2	6.0–8.0	0.51–0.57	0.45–0.50	8	—	—	2.0	15	—
—	3.8	4.49	0.85	0.75	—	—	—	—	—	—
19.0	0	6	0.36	0.32	—	—	—	—	—	—
0	0	35	—	—	—	0	0	0	0	0
27.0	—	11.4	1.07	0.94	0	0.08	100	0.8	1.0	35
—	—	—	1.06	0.93	0	—	—	—	—	—
18.0	—	—	0.46	0.40	0	—	—	—	—	—
0	0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0	0	0
0	0	0	—	—	0	0	0	0	0	0

	Crude protein (g/kg)	MADC		Oil (g/kg)	Crude fibre (g/kg)	MAD fibre (g/kg)	NDF (g/kg)	Ash (g/kg)	Ca (g/kg)	P (g/kg)
		(g/kg DM)	(88% DM)							
Pasture:										
1. First growth	167	103	91	38	176	194	400	97	5.3	3.1
2. Second growth	180	130 to 92	114 to 81	35	195	215	425	90	5.3	1.9
Pure clover	200	112	99	31	176	229	275	97	15.8	1.9
Pure grass	176	98	86	13	264	290	450	85	5.7	1.8
3. Blooming	—									
Pure clover	150	96	84	26	211	308	340	105	14.1	2.0
Pure grass	79	40	35	13	264	290	570	92	3.1	1.8
4. Winter after close grazing until July, and free growth from July to December	136			26	194			70		

* Contains 30 g Na/kg.

Exp., expeller; extr., extracted; MAD, modified acid detergent.

K (g/kg)	Lysine (g/kg)	DE (MJ/kg)	UFC		α -Toco- pherol (mg/kg)	Free folic acid (mg/kg)	Available biotin (μ g/kg)	Thiamin (mg/kg)	Riboflavin (mg/kg)	Pantothenic acid (mg/kg)
			(g/kg DM)	(88% DM)						
26	5.0	7.4–9.3	0.88	0.77						
21		9.6	0.79	0.69						
18		9.0	0.8	0.7						
21		9.5	0.8	0.7						
17		8.8	0.7	0.6						
15		8.4	0.67	0.59						
		6.6				—				

(b) Forages

	Crude protein (g/kg)	MADC (g/kg DM)	Oil (g/kg)	Crude fibre (g/kg)	MAD fibre (g/kg)	NDF (g/kg)	Ash (g/kg)	Ca (g/kg)	P (g/kg)	K (g/kg)	Lysine (g/kg)	DE (MJ/kg)	UFC (g/kg DM)	UFC (88% DM)	pH	NH ₃ -N as % of total N
Pasture:																
(1) First growth	167		38	176	194	400	97	5.3	3.1	26	5.0	7.4–9.3			—	—
(2) Second growth	180		35	195	215	425	90	5.3	1.9	21	—	9.6	0.79	0.69	—	—
Pure clover	200		31	176	229	275	97	15.8	1.9	18	—	9.0	0.8	0.7	—	—
Pure grass	176		13	264	290	450	85	5.7	1.8	21	—	10.0	0.8	0.7	—	—
(3) Blooming																
Pure clover	150		26	211	308	340	105	14.1	2.0	17	—	8.8	0.7	0.6	—	—
Pure grass	79		13	264	290	570	92	3.1	1.8	15	—	8.4	0.67	0.59	—	—
(4) Winter after close grazing until July, and free growth July–December	136		26	194	—	—	70	—	—	—	—	6.6			—	—
Clamp silage	105–160	40–60	28	299	334		62	4.8–5.5	2.6–3.3	15	—	9.2–11.9	0.6–0.68	0.53–0.6	4.2	12.4
Big-bale silage	98–110	36–55	35	232	273		62	4.0–4.6	2.8–3.3	—	—	8.7–9.8	0.65–0.72	0.57–0.63	5.1	8.9
Grass haylage	62	—	—	—	—	—	—	5.1	1.8	—	—	8.0	—	—	5.5–6.2	—
Lucerne hay mid-bloom	150–160	84	17	270	350	415	70	11.4	1.9	16	—	7.6	0.5–0.63	0.44–0.55	—	—

(c) Some factors necessary for the estimation of km , used in the calculation of UFC values and the estimation of UFC values from chemical composition of feeds. Gross energy (GE, kJ/g) and km % of nutrients

	GE	km
glucose (GL)	15.65	85
acetate (C ₂)	14.60	
propionate (C ₃)	20.76	
butyrate (C ₄)	24.94	
amino acid (AA)	23.44	70*
long chain fatty acids (LCFA)	39.76	80

* amino acid ME

So

$$km = 0.85E_{GL} + 0.80E_{LCFA} + 0.70E_{AA} + (0.63 \text{ to } 0.68) E_{VFA} - 0.14 (76.4 - ED),$$

where E is the % of absorbed energy from glucose or lactate (GL) etc., ED is energy digestibility (%) of the feed and the last term of the equation corresponds to the cost of eating, a term not included for concentrate feeds. The percentage of absorbed energy from a typical horse diet of roughage and concentrate is represented by 9–41% GL, 45–82% VFA, 7–17% AA and 2–6% LCFA (Vermorel *et al.* 1997).

(d) Variations in molar proportions, energy content (E, kJ/g VFA mixture) and efficiency (km) of E utilization of VFA mixtures absorbed from the colon of the horse receiving diets of three crude fibre concentrations (Vermorel & Martin-Rosset 1997):

Crude fibre % DM:	15	20	30
C ₂ %*	65	68	73
C ₃ %	21	19	16
C ₄ %	14	13	11
E, kJ/g	17.91	17.70	17.24
km %	66.6	65.8	64.5

* The molar proportion of acetate (C₂) in the VFA mix increases with increasing dietary crude fibre (CF), C₂ % = 0.54 CF (% DM) + 57. The energy expended in mastication also increases with increasing dietary crude fibre, $\Delta km = -0.20 \text{ CF\%} + 2.50$, so the above km values decrease further from 15% to 30% CF. Both the heat of

fermentation and the energy wasted in metabolism are greater with C₂ metabolism than with the other VFA. Thus increasing fibre is associated with increased waste heat production and poor meadow hay has a *km* % of only 61, whereas that of maize is 80.

(e) Prediction of UFC value per kg dry matter (DM) of forages and concentrates from their cytoplasmic carbohydrate (CC), crude protein (CP), crude fibre (CF) and digestible organic matter (DOM), kg/kg DM, or digestible energy (DE, MJ/kg DM) content (Vermorel & Martin-Rosset 1997).

(i) Forages

$$\text{UFC} = -0.124 + 0.254\text{CC} + 1.330\text{DOM}, \text{RSD } 0.012, \text{R}^2 \text{ } 0.988.$$

$$\text{UFC} = -0.056 + 0.562\text{CC} + 0.0619\text{DE}, \text{RSD } 0.007, \text{R}^2 \text{ } 0.996.$$

(ii) Concentrates

$$\text{UFC} = -0.134 + 0.274\text{CF} - 0.362\text{CP} + 0.316\text{CC} + 0.0755\text{DE}, \text{RSD } 0.017, \text{R}^2 \text{ } 0.995.$$

NB: See Martin-Rosset (1996c) for CC values, i.e. water-soluble carbohydrates, of feeds and reference to a comprehensive data source.

(iii) Compound feeds

$$\text{UFCo} = 1.333 - 1.684\text{ADFo} - 0.096\text{CPo}, \text{RSD } 0.060, \text{R}^2 \text{ } 0.958.$$

NB: Martin-Rosset *et al.* (1996c) define UFCo as a UFC per kg organic matter to allow for the high mineral content of some compound feeds. UFCo values of compound feeds must be increased by 0.02 UFCo for each 1% ether extract above 3.5% of feed.

(iv) Organic matter digestibility of forages (OMD %)

Three alternative methods are proposed for the determination of OMD % of horses, of which the second and third are the preferred methods.

(1) $\text{OMD \%} = 67.78 + 0.07088\text{CP} - 0.000045\text{NDF}^2 - 0.12180\text{ADL}$, RSD 2.5, R² 0.878, Martin-Rosset *et al.* (1996a); where NDF is neutral detergent fibre, g/kg DM and ADL is acid detergent lignin, g/kg DM.

(2) OMD % in horses predicted by near infrared spectrophotometry (NIRS); RSD 1.80, R² 0.93, Andrieu *et al.* (1996).

(3) $\text{OMD \%} = -29.38 + \text{di} + 2.3032\text{CDMD} - 0.01384\text{CDMD}^2$, RSD 1.90, R² 0.927, Martin-Rosset (1996b); where di = +4.12 for green forages, di = 0 for grass hays and di = -2.61 for legume hays; CDMD is the pepsin cellulase degradability (%) of DM.

Appendix D

Estimates of Base Excess of a Diet and of Blood Plasma

ESTIMATE OF BE OF A DIET FROM ITS FIXED ION CONTENT

(See Chapter 9 for further details.)

$$(\text{Cations} - \text{anions})_{\text{absorbed}} - (\text{cations} - \text{anions})_{\text{excreted in urine}} - \text{H}^+ \text{ endogenous} = \text{BE} \quad (1)$$

Account here is taken only of fixed ions absorbed from the diet and Eq. (2) shows the principal ones involved. (NB: fixed ions are those that cannot be degraded by metabolism.)

$$(\text{Cations} - \text{anions})_{\text{absorbed}} = \text{mEq}(0.95 \text{ Na} + 0.95 \text{ K} + 0.5 * \text{Ca} + 0.5 \text{ Mg}) - \text{mEq}(0.95 \text{ Cl} + 0.95 \text{ S} + 0.5 * \text{P}) \quad (2)$$

* Approximate values which will be inversely related to dietary concentration.

In order to avoid a degree of arbitrariness a simplified balance of ions has been proposed and these are shown in Eq. (3) in relation to their optimum range in the diet of a horse in light work.

$$(\text{Na} + \text{K} - \text{Cl})_{\text{absorbed}} = 200\text{--}300 \text{ mEq/kg diet} \quad (3)$$

(Note: $\text{Na}^+ + \text{K}^+ = 95\%$ of all cations in extracellular fluid and $\text{Cl}^- + \text{HCO}_3^- = 85\%$ of all anions.)

ESTIMATE OF BE OF BLOOD PLASMA FROM ITS BICARBONATE CONCENTRATION

Plasma bicarbonate (HCO_3^-) (mEq/l) at pH 7.4:

$$\equiv [\text{HCO}_3^-]_{\text{measured}} - 10(7.4 - \text{pH measured})$$

BE of plasma at pH 7.4:

$$\cong [\text{HCO}_3^-] \text{ at pH } 7.4 - 24$$

(Normal bicarbonate of venous blood at pH 7.4 = 24 mEq/l.)

If the venous plasma of a horse was found to have a pH of 7 and $[\text{HCO}_3^-]$ was 30 mEq/l then $[\text{HCO}_3^-]$ at pH 7.4 would

$$= 30 - 10(7.4 - 7)$$

$$= 26 \text{ mEq/l}$$

Therefore

$$\text{BE} = 26 - 24$$

$$\cong 2 \text{ mEq/l}$$

NB: other organic acid anions could also be included in a BE calculation.

Glossary

Terms in the definitions that are themselves entries are shown in bold face.

aboral	Away or remote from the mouth.
acidaemia	An increased hydrogen ion concentration (acidity) and lowered blood bicarbonate, or decreased pH of the blood (see p. 557).
acidosis	See acidaemia .
ACTH	Adrenocorticotrophic hormone (corticotropin) is secreted by the anterior pituitary gland, so controlling cortisol secretion by the adrenal cortex. The release of ACTH is in turn controlled by corticotropin-releasing hormone (CRH) secreted in the hypothalamus.
acute	Applied to a metabolic upset, or a disease, which progresses rapidly to a climax followed by death or rapid recovery. Contrasts with a chronic condition or disease.
acute-phase proteins	Specific serum proteins which increase rapidly in concentration (up to 100-fold) following infection, e.g. C-reactive protein, and which increase during inflammatory response, e.g. caeruloplasmin (ferroxidase), which also transports Cu (contains about 3% of the body's Cu).
adipose tissue	The cells of this tissue readily store fat which is drawn on as a source of energy, especially when the blood levels of glucose and VFAs are low.
<i>ad libitum</i> feeding	A system in which feed supply is unrestricted at all times except during exercise. However, usually applies only to growing horses.
adrenal glands	A pair of ductless glands, one situated near each kidney, and consisting of an internal medulla, which secretes the hormones epinephrine and norepinephrine (adrenaline and noradrenaline), and an external cortex, which secretes corticosterone, cortisol (glucocorticoids) and aldosterone.

aerobic	In aerobic respiration, energy-yielding nutrients are broken down with the consumption of dissolved oxygen that has reached the tissue cells from the lungs. In anaerobic respiration the breakdown of these nutrients is incomplete, it yields less energy and occurs in the absence of oxygen.
afferent	Afferent nerve fibres conduct impulses centripetally, e.g. from sense organs to the central nervous system.
agalactia	Failure of the secretion of milk.
agglutination	The clumping together of particulate antigen (foreign substance) in the presence of homologous antibody (defence substance).
air dry	Under UK regulations, feed that has been allowed to dry without heating in the air contains 100–140 g moisture/kg.
aldosterone	The main mineralocorticoid hormone secreted by the adrenal cortex, promoting the reabsorption of Na and therefore water by the kidney tubules.
alfalfa	See lucerne .
alimentary canal/tract	See gastrointestinal tract . In addition, it includes the buccal cavity and oesophagus.
alkalaemia	A decreased hydrogen ion concentration, or elevated pH , of the blood, irrespective of changes in blood bicarbonate. Normal arterial blood pH is 7.5 (see p. 557).
allergy	A condition of exaggerated susceptibility, or sensitivity, to a specific substance, usually, but not necessarily, containing a specific protein. Exposure, especially to large amounts of the allergen, through inhalation, ingestion, injection or even by skin contact, may cause respiratory difficulties, sneezing, a skin rash or diarrhoea of increasing severity through repeated contact.
ALT (also GPT)	Alanine aminotransferase (EC 2.6.1.2), formerly called glutamic pyruvatetransaminase. The activity of this enzyme in the blood plasma shows a similar reaction to that of AST , particularly in respect of exercise and muscle damage.
amino acids	These N-containing compounds are the building-blocks of proteins. There are some 25 different kinds, ten of which are known as dietary indispensable (essential) nutrients, the most critical of which is lysine.
α-amylase	An important digestive enzyme in the digestion of starch and other polysaccharides containing three or more α -1,4-linked D-glucose units. It hydrolyses the α -1,4-glucan links.
anabolism	The process of synthesis of complex organic molecules in the body from simpler precursors (cf. catabolism).
anaemia	A condition in which there is a reduced number of red cells and/or a reduced haemoglobin content of the blood. The volume of packed red cells is reduced when the equilibrium

	between blood loss, through bleeding or destruction, and blood production is disturbed.
analgesic	A pain-relieving substance.
angiogenesis	The development of vessels; in the text it refers to blood vessels.
anion gap	The anion gap is the difference between measured cations and measured anions. It quantitatively reflects those ions that are unmeasured because of the principle of electroneutrality, in which the sum of all anions and all cations must be equal in both plasma and urine. $[\text{Na}^+] + [\text{K}^+] + \text{UC} = ([\text{HCO}_3^-] + [\text{Cl}^-]) + \text{UA}, \text{ so}$ $[\text{UA}] - [\text{UC}] = [\text{Na}^+] + [\text{K}^+] - ([\text{HCO}_3^-] + [\text{Cl}^-]) = \text{Anion gap},$ where UA = unmeasured anions and UC = unmeasured cations, both measured as mEq/l. It is used to distinguish titration and secretion forms of metabolic acidosis. The gap increases as the pH falls in the titration form (e.g. increased lactic acid), whereas the gap is normal in secretion form. In acid urine (pH < 6.5) the bicarbonate concentration is very low and negligible and there is normally a negative correlation between anion gap and urinary ammonium ion.
anorexia	Lack, or loss, of appetite for feed.
anthelmintic	A substance used to destroy parasitic worms.
antibiotic	A chemical substance produced by and obtained from living cells, especially of lower plants such as moulds, yeasts or bacteria, that is antagonistic to, or destroys, some other form of life. It may be so used to destroy infectious organisms.
antibody	A specific substance (immunoglobulin) found in the blood, or in certain secretions, in response to the antigenic stimulus of bacteria, viruses, worm parasites and certain other foreign substances. An antibody has a specific amino acid sequence and can combine specifically with the inducing foreign entity (antigen), helping to inactivate it.
antigen	Any substance that is capable under appropriate conditions of inducing the formation of antibodies and of reacting specifically with those antibodies.
antihistamine	A drug that counteracts the effects of histamine or certain other amines that cause inflammation.
antitoxin	A substance found in blood serum or other body fluid that is antagonistic to a particular toxin . For therapeutic or protective use it may be injected into horses to neutralize the toxin of a particular disease, but as it does not stimulate the horse to produce its own antitoxin, its (passive) effects may last for only a few weeks.
arrhythmia	Any variation from the normal rhythm of the heart beat.

artery	A vessel containing smooth muscle through which blood passes away from the heart to the various parts of the body.
ascarids	Roundworms. A group of large parasitic intestinal parasites in the phylum Nematoda. They are 15–20 cm long, white and infest primarily young horses, as those over three to four years of age have usually developed considerable immunity (for life cycle see Chapter 11). Large numbers of adult worms in the intestines can cause impactions, intestinal perforations and colic.
ascites	Effusion and accumulation of serous fluid in the abdominal cavity.
ash	The ash content of feed is determined by ignition of a known weight of it at 500°C until the carbon has been removed. The residue represents approximately the inorganic constituents of the feed – principally Ca, K, Mg, Na, S, P and Cl. Some feeds, particularly those contaminated with soil, may contain a significant amount of silica.
AST (also AAT and GOT)	Aspartate aminotransferase (EC 2.6.1.1), formerly called glutamic oxaloacetic transaminase. This enzyme is released into the blood following damage to liver or muscle cells so that the blood level rises sharply. Plasma activity of the enzyme usually increases after hard exercise. The normal maximum blood plasma level in adult horses is 250 iu/l.
ataxia	Failure of muscular coordination, or irregularity of muscular action, resulting in a staggering gait. It may result particularly from exhaustion or from pathological change in the nerves.
ATP	Adenosine triphosphate mediates the transfer of energy from the breakdown of glucose and fatty acids (exergonic reactions), for the synthetic processes of growth, milk secretion, etc. (endergonic reactions) and for muscular action. ATP is split by the enzyme ATPase, with the liberation of inorganic phosphate.
autogenous	Self-generated. It refers particularly in the text to antibodies produced by the dam to blood proteins of the foetus that happen to be circulating in her blood. If colostrum is then taken by the foal within the first 12 hours, there will be an antibody reaction with the foal's blood proteins.
autonomic nervous system	This is the self-controlling part of the nervous system – it is not subject to direct influence by the conscious brain. The sympathetic system (thoracolumbar outflow) and the parasympathetic system (craniosacral flow) have largely antagonistic actions. The combined system is of importance in regulating the activities of many of the glands, the smooth

	musculature of the GI tract and elsewhere, and the heart and blood vessels.
availability of minerals	The proportion of a nutrient mineral supplied in the feed which, at a stated level of inclusion and level of feeding, can be absorbed and utilized by the horse to meet its net requirement. It is not necessarily synonymous with true digestibility.
azotaemia	An excess of urea and other nitrogenous compounds in the blood.
azoturia	An excess of nitrogenous compounds in the urine. Considered synonymous with exertion myopathy. It frequently occurs within a short interval of beginning exercise after a rest of two or three days. It is characterized by a reluctance to move and muscular spasms and excessive lactic acid accumulates in the muscles. The muscles of the hindquarters become tense and there is a tendency to knuckle-over at the fetlocks. At some stage the horse will pass quantities of urine from light-Burgundy-wine to dark-coffee colour. Sometimes the urine is retained and requires relief with a catheter.
base excess and base deficit	Base excess is defined as the titratable base using a strong acid to titrate to a pH of 7.4 at a P_{CO_2} of 40 mm Hg at 37°C. Base excess is not a measure of the total buffering power and it may be derived, under certain circumstances, from pH and bicarbonate alone. Base deficit is negative base excess and is defined as the titratable acid using a strong base, also to titrate to pH 7.4 (NB: normal pH: arterial 7.347–7.475; venous 7.345–7.433).
β-carotene	See carotene .
big-head	A condition seen in horses and ponies given a ration based on cereals, bran and poor hay without adequate mineral supplementation. The bones of the upper jaw and face are particularly enlarged owing to a replacement of their normal mineralized structure by fibrous connective tissue.
bile duct	The duct carrying bile, synthesized by the liver, to the duodenum, where the bile facilitates emulsification of fats and contributes to the production of an alkaline reaction of the intestinal contents.
biological values	The amount of N retained by an animal per unit of N absorbed from the GI tract and from a given feed protein.
biuret	A simple organic compound containing three N atoms, which has been put forward as a source of dietary NPN. The structurally related compound urea contains only two N atoms per molecule.

bleeders	Broken blood vessels. This usually refers to the loss of blood through the nose after hard exercise. Blood is lost from small broken blood vessels in the lungs, or nasal passages, of about half the population of healthy TBs during hard exercise and the condition requires no treatment unless large blood vessels are involved.
blood counts	These normally include the numbers of red blood cells (erythrocytes) per unit volume, the packed cell volume (haematocrit), or proportion of red blood cells by volume in the total blood measured after the blood has been centrifuged, and the haemoglobin content of the total blood. Three other characteristics of the red cells (MCV, mean corpuscular volume; MCH, mean corpuscular haemoglobin and MCHC, mean corpuscular haemoglobin concentration) are calculated from these basic data. The numbers of white blood cells (leucocytes) per unit volume and the differential count (proportion of each type of white cell) may also be measured.
borborygmus	Rumbling noise caused by the propulsion of gas through the intestines.
bot fly	The larvae, or maggots, of <i>Gastrophilus</i> spp. cause chronic gastritis and loss of condition in grazing animals. <i>Gastrophilus intestinalis</i> lays eggs of pin-head size on the horse's hair in the summer. When the horse licks itself, the eggs are taken into the mouth and hatch there or in the stomach. On rare occasions they cause perforation of the stomach and death (see Chapter 11).
botulism	A rapidly fatal motor paralysis caused by the ingestion of the toxin of <i>Clostridium botulinum</i> , a spore-forming anaerobic bacterium, which proliferates in decomposing animal tissue and sometimes in plant material. The toxin (a di-chain protein, MW 140000) is the most neurotoxic substance known. In the text (Chapter 10), botulism is referred to in cases in which horses have consumed silage that has been subject to an abnormal fermentation. The toxin seems to inhibit irreversibly the release of acetylcholine from peripheral nerves, and so impedes neuromuscular transmission. A flaccid descending paralysis develops.
brachygnathia	Abnormally short lower jaw bone, creating difficulties, for example, in grazing.
bradycardia	Slow heart beat.
broken wind, heaves	These are outdated expressions applied to long-standing respiratory diseases in which a double expiratory effort is a feature. The causes include bacteria, viruses and, rarely,

	lung tumours. A common cause is an allergic reaction, and that related to the inhalation of spores and particles of the moulds <i>Micropolyspora faeni</i> and <i>Aspergillus fumigatus</i> is discussed in Chapter 11.
bruxism	Rhythmic or spasmodic grinding of teeth.
buccal cavity	The cavity of the mouth between the cheeks bounded at one end by the lips and at the other by the pharynx.
buffers	Substances that in aqueous solution increase the amount of acid or alkali that may be added without changing the pH, or degree of acidity or alkalinity. In general, a buffer is made up of two components: (1) a weak acid, e.g. H_2CO_3 , and (2) its corresponding base, HCO_3^- . Arterial blood is well buffered around a pH of 7.5.
cachexia	A profound and marked state of constitutional disorder; general ill health and malnutrition.
caecum	A great cul-de-sac intercalated between the small intestine and the colon. In the adult horse it is about 1.25 m long, with a capacity of 25–30 l (see Chapter 1).
calcitonin	A hormone synthesized by the parafollicular cells of the thyroid gland . It is secreted when the serum concentration of Ca ions rises, promoting the deposition of Ca in bones, and so counteracting the action of parathyroid hormone .
calculi	Urinary calculi consist of accumulations of mineral substances in the urinary tract. They form in the bladder, less frequently in the urethra and rarely in the kidneys. They are commonly rough and yellow-brown and are composed of calcium carbonate. These are seen more commonly in horses on high-roughage diets or on pasture. The less common phosphate calculi are smooth and white and occur with high-cereal rations. A low intake of water may predispose the horse to calculi and the signs include difficulty in urination and incontinence.
calorie	A unit of energy, being the amount of heat required to raise 1 g water 1°C. 1000 calories = 1 kilocalorie (kcal). The joule (J) has now been adopted as the unit of energy in nutrition; 4.184 kJ = 1 kcal.
carbohydrates	Compounds composed of carbon, hydrogen and oxygen and including the sugars, starches and other storage carbohydrates and the structural (fibre) carbohydrates – cellulose and hemicelluloses; also pectins, gums and mucilages. Lignin is included in the structural fibre but is not strictly a carbohydrate.
cardiac output	The volume of blood expelled by each ventricle per minute. The stroke volume is the volume of blood discharged by

	each ventricle at each beat (i.e. the cardiac output divided by the heart rate). The cardiac output of the left ventricle after birth is 2–8% higher than that of the right, so the value quoted represents the mean of these two.
carotene	Green plants contain a number of yellow carotenoid pigments, the most important of which are α -, β - and γ -carotenes and hydroxy- β -carotene. The most potent of these, β -carotene, is converted to vitamin A by the intestinal wall.
carpus	The 'knee' of the forelimb of the horse between the radius above and metacarpus below.
catabolism	The breaking down of tissue nutrients and components to less complex molecules (cf. anabolism).
catecholamines	A group of similar compounds having sympathomimetic action. They include epinephrine, norepinephrine and dopamine. The latter two serve as neurotransmitters and the former two are hypertensive, stimulating smooth muscles.
cathartic	A purgative, or medicine, that quickens the evacuation of the bowels (intestines).
cation	The positively charged elements of electrolytes, which include all metals and hydrogen ions.
cellulolysis	The breaking down, or digestion, of cellulose. Horses and other mammals depend on certain bacteria in their intestinal tracts to carry this out as they do not secrete enzymes capable of it.
cellulose	A structural carbohydrate (fibre) of plant cells.
choke	Obstruction to the passage of food through the pharynx and oesophagus, either partial or complete. This is frequently caused by a mass of dry impacted feed.
cholestasis	Suppression of bile flow. This may be unusually caused by gallstones and is then termed <i>cholelithiasis</i> .
chondrocyte	A mature cartilage cell embedded within the cartilage matrix.
chronic	Long-continued; the opposite of acute .
CK (or CPK)	Creatine kinase (EC 2.7.3.2). This enzyme is measured as an indicator of muscle damage. The normal maximum plasma level in adult horses is 105 iu/l.
coenzyme	A non-protein organic compound, which may be a vitamin, the presence of which is required by an enzyme for the catalysis of the particular reaction to occur.
coldblooded horse	In Europe two types of horse evolved – the light short-legged Celtic pony and the Great Horse of the Middle Ages (the large powerful but slow heavy horse). Present-day

	breeds whose major blood lines derive from either of these types are termed 'coldblooded' (cf. hotblooded horse).
colic	Abdominal pain. Where this originates from part of the GI tract it is termed 'true colic', but where it derives from one of the other vital organs or muscles it is defined as 'false colic'.
colon	Made up of the great colon, which originates from the caecocolic orifice and terminates where it joins the small colon, and the latter, which continues to the rectum. In the adult horse the great colon is 3–3.7m long with an average diameter of 20–25cm, whereas the small colon has a diameter of 7.5–10cm and a length of about 3.5m.
colostrum	Secreted by the mammary gland of the mare shortly before foaling (parturition) and for about the first 24 hours after the birth of the foal. It is rich in γ-globulins , which comprise the antibodies that the foal absorbs undigested into its blood during the first 12–18 hours of life, providing it with a measure of protection from disease.
compounded feeds	Balanced mixtures of ground or otherwise processed feedstuffs, to which appropriate supplements of vitamins, minerals and trace elements have been added.
concentrates	The portion of the horse's ration, or a feedstuff that is rich in starch, protein or both, and that contains less than 15–17% crude fibre .
conductance	Capacity for conducting and, in reference to this text, heat transfer from one medium, or body, to another. The unit of electrical conductance is the mho.
contagious	Transmissible from one horse to another.
contracted tendons	Hyperflexion or flexural deformity of limbs. The condition in foals ranges from uprightness of hind or forelegs to knuckling over at the fetlock and/or inability to extend knee joints. Not uncommon in TB foals. Correction of slight abnormalities may include the fitting of boots and in more severe cases severance of fibres of the flexor muscles and tendons or desmotomy of the superior check ligament.
convulsions	A violent involuntary contraction, or series of contractions, of the voluntary (skeletal) muscles.
coprophagy	The eating of faeces by an animal. Within three weeks of birth, foals will eat their dam's faeces and thereby acquire the species of bacteria and protozoa necessary for the rapid development of a normal microbial population in their GI tract so that invasion by harmful microorganisms is partially inhibited.

cornea	The transparent structure that forms the anterior part of the eyeball.
coronary band (coronary matrix)	Runs around the horse's foot just below the hair line and forms part of the sensitive structures from which the wall grows. A permanent defect in the hoof wall usually follows injury to the coronary band.
corpus luteum	'Yellow body.' A yellow glandular mass in the ovary, formed by an ovarian follicle that has matured and discharged its ovum. It contains carotenoids and secretes progesterone.
cortical bone	Compact bone forming the cylinder of the shaft, or diaphysis of long bones, and resulting mainly from periosteal ossification.
corticosteroids	Comprise the natural glucocorticoids, cortisone and hydrocortisone hormones secreted by the adrenal cortex and synthetic equivalents, e.g. prednisone, prednisolone and fluoroprednisolone, used in the treatment of inflammation, shock, stress and, in other animals, ketosis.
CP	Creatine phosphate (phosphocreatine). The fixation of energy in the form of ATP is a transitory phenomenon, and any energy produced in excess of immediate requirements is stored more permanently in compounds such as muscle phosphocreatine. As ATP becomes depleted, more is generated from phosphocreatine by a reverse reaction.
creatinine	The normal excretory breakdown product of muscle creatine found in horses' urine. As the quantity produced daily is relatively constant for a particular horse, being proportional to muscle mass, its concentration in urine is used in the assessment of the level of other substances excreted in urine (see Chapter 12). Blood levels of creatinine rise dramatically following renal failure.
creatine kinase	See CK .
creep feed	A feed, normally dry pellets, offered to nursing foals behind a barrier, which does not allow the mare access, but permits the foal to enter.
cribbing (aerophagia)	An outdated expression for wind-sucking , which is a vice of domesticated horses and ponies. This consists of the habitual swallowing of air while the animal bites or pulls down with its upper incisor teeth on some solid object such as a fencing rail or gate. The neck is slightly arched and gulps of air are swallowed into the stomach with emission of a grunt. The term 'wind-sucking' is also unfortunately used to describe mares that aspirate air, and frequently faecal material, into the vagina. This is rectified by Caslick's operation.
crimping	A term used for the pressing of cereal grains between corru-

	gated rollers to rupture the kernels and to increase digestibility slightly.
croup	That part of the horse's hindquarters lying immediately behind the loins. The 'point of the croup' is its highest part and corresponds to the internal angles of the ilia.
crude fibre	The feed residue identified after subjecting the residual feed from ether extraction to successive treatments with boiling acid and alkali of defined concentrations. The crude fibre contains cellulose, hemicellulose and lignin, but it is not an accurate measure because it underestimates the structural components of vegetative matter (see NFE).
curb	A swelling about 100 cm below the point of the hock, owing to undue strain causing a sprain of the calcaneocuboid ligament, or of the superficial flexor tendon.
cyclic AMP	An intracellular hormonal mediator formed from ATP under the influence of stimulating hormone.
DE	Digestible energy. The gross energy (or heat of combustion) of a feed minus the gross energy of the corresponding faeces, expressed as MJ or kJ/kg total feed. Synonymous with apparent digestible energy.
deamination	When amino acids are present in excess of needs for tissue protein synthesis, or when the horse is forced to catabolize tissue to maintain essential functions, amino acids may be degraded to provide energy. This occurs mainly in the liver and to some extent in the kidneys. The first step in the oxidative degradation of amino acids is the removal of the amino group, a process called deamination. This group is then either transferred to a keto acid to produce another amino acid or is incorporated into urea.
decarboxylation	In the text, this term is principally restricted to reactions of amino acids. Bacteria – for example, in the intestinal tract and in silage during the early stages of fermentation of the clamp – elaborate enzymes (called carboxylases), which act upon amino acids to yield amines and carbon dioxide. This implies a loss of dietary protein value and the amines, including histamines and tryptamine, may have toxic effects following absorption.
dehydrated	Feed from which most of the moisture has been removed. This extends shelf life and greatly retards the rate of, or inhibits, mould spoilage.
dialysis	The process of separating crystalloids from colloids in solution by the difference in their rates of diffusion through a semipermeable membrane; colloids pass very slowly or not at all.

diaphragm	A thin muscular partition or membrane that separates the thorax (chest cavity) from the abdomen. During its contraction air is drawn into the lungs and when it relaxes air is expelled.
diaphysis	The shank of a long bone between the ends, or epiphyses, which are usually wider and articular.
dicoumarol	An anticoagulant with similar properties to warfarin except that its action has a slower onset, a longer duration and a less predictable response. When ribbed melilot or yellow sweet clover (<i>Melilotus officinalis</i>) or white melilot or white sweet clover (<i>M. alba</i>) plants are damaged by weather, badly harvested, or when, as hay, they become mouldy, coumarin contained in the sweet clover is broken down to dicoumarol. Both yellow and white sweet clover are found in pastures in the UK, and white sweet clover is grown as a forage crop in North America and the former USSR.
digestible energy	See DE .
dispensable amino acids	Amino acids that are synthesized in the tissues of horses, and/or are made available from synthesis by gut microorganisms, in amounts sufficient to meet tissue requirements of horses without a dietary source.
distal	Remote, farthest from the centre, or origin, as opposed to proximal .
diuresis	Increased secretion of urine. A diuretic drug induces diuresis.
dopamine	A neurotransmitter and an intermediate in the synthesis of norepinephrine.
duodenum	The first (proximal) part of the small intestine and connected to the stomach. In the adult horse it is 1 m long with a diameter of 5–10 cm.
dyschondroplasia	Disordered, or abnormal, cartilage formation.
dysphagia	Difficult in swallowing.
dysplasia	Abnormality in the development of cells.
dyspnoea	Difficuly or laboured breathing.
dystrophy	Faulty nutrition. Dystrophy of muscles causes their atrophy and degeneration.
electrolytes	Substances that in water solution break up into particles carrying electrical charges. The principal electrolytes from a nutritional point of view are Na^+ , K^+ , Cl^- , HCO_3^- , Ca^{2+} , Mg^{2+} and HPO_4^{2-} .
emphysema	A swelling, or inflation, of the chest which is caused principally by the presence of air in the intra-alveolar tissue of the lungs following rupture of the alveoli.
endochondral ossification	Bone formation within cartilage, cf. periosteal ossification.

endocrine gland, hormones and related secretions	An endocrine gland secretes a specific substance (hormone) that is released directly into the circulatory system (blood, lymph or neurosecretory channels) and which influences metabolism at sites in the body distant from the gland. Two other types of hormonal secretion are: an <i>autocrine</i> secretion, which influences the function of the cells that produce it; and a <i>paracrine</i> secretion, which is produced by one type of endocrine cell and influences the secretions of nearby cells that are not its target. An <i>exocrine</i> gland is one that secretes outwardly via a duct opening onto an internal or external epithelial surface.
endogenous	Arising from within the horse, i.e. excluding the lumen of the GI tract.
endotoxaemia	Presence in the blood of endotoxins, which are non-protein, lipopolysaccharide fragments of the cell wall of Gram-negative bacteria. Per milligram they are much less toxic than are exotoxins, but appear to play a crucial role in certain diet-related disorders of bowel origin, including forms of colic and founder .
enema	A fluid for injection into the rectum or small colon for cathartic or diagnostic purposes.
enterotoxaemia	Presence of toxins in the blood, produced and secreted in the intestines by certain bacteria, e.g. those of <i>Clostridium perfringens</i> , which are referred to in the text (Chapter 11). Enterotoxin produced in large quantities by this organism is also specific for cells of the intestinal mucosa and causes enteritis. This anaerobic organism is found in soil and the consumption of relatively small numbers of the spores in herbage is usually without remarkable effect (see also botulism).
epinephrine	See catecholamines .
epiphysis	A head of a long bone joined to the shaft, or diaphysis, during growth by a cartilaginous growth plate – a metaphysis.
epiphysitis	Inflammation of an epiphysis or of the cartilage that separates it from the shaft during growth.
epistaxis	A nose bleed. Blood may be present in the nostrils through being coughed up from broken blood vessels in the lung. Small losses can be a normal phenomenon after a race, but persistent losses may occur in chronic bronchitis.
epithelial cells	All the body surfaces, including the external surface of the skin, the internal surfaces of the digestive, respiratory and genito-urinary tracts, the inner coats of vessels and ducts of all secreting and excreting glands are covered by one or more layers of cells called epithelium or epithelial cells.

ergot	A fungus that infects and finally replaces the seed of a cereal or grass, especially the sclerotium of <i>Claviceps purpurea</i> (ergot of rye). This ergot is small, hard, black and resembles mouse droppings. Ergot contracts the arteriolar and other unstriated muscle fibres. Its toxins are used to arrest haemorrhage after parturition and internal injury. The persistent consumption of ergot of rye sufficiently decreases blood flow to cause gangrene (typically of the ears, tail and legs). Several ergot toxins cause abortions. After eating large amounts of ergotized hay, horses become dull and listless, a cold sweat breaks out on the neck and flanks, the breathing is slow and deep, the body temperature is subnormal and the pulse weak. Death occurs during a deep coma within the first 24 hours. Lesser amounts over a longer period may cause diarrhoea, colic, trembling and loss of condition.
erythrocytes	See red blood cells .
essential amino acids	See indispensable amino acids .
ether extract	Chemical substances that are soluble in, and extracted by, ether. Whether diethyl ether, or 40:60 petroleum ether, is used should always be specified.
exertion myopathy	Azoturia myohaemoglobinuria. An acute condition in which affected muscles, especially of the hindquarters, become hard to the touch, and the hind limbs rapidly become stiff and weak or staggering. There is a tendency to knuckle-over at the fetlocks. The risk is greatest in horses that have been in continuous work, abruptly rested for a few days on full feed and then returned to work.
expansion, extrusion	This relies on the cooking effects of super-heated steam injected into a slurry compressed against a die face by a revolving worm and the subsequent rapid fall in pressure during extrusion. Material is subjected to a temperature of around 120°C for about a minute.
extracellular space	The fluid space, or volume, within the body that is external to the cells.
fasciculation	Local contraction, or bundling, of muscles.
fatty acids	Composed of a hydrocarbon chain of one to more than twenty units attached to a carboxyl group. In the formation of storage fats, fatty acids are neutralized by the trihydric alcohol glycerol. Both neutral fat and fatty acids circulate in the blood (see also free fatty acids and volatile fatty acids).
feed	That which is given to the animal to consume, whether a single feedingstuff or a mixture, but excluding water. Not synonymous with ration .

fermentation	Decomposition of organic substances by microorganisms. In the horse's GI tract this refers especially to bacteria, yeasts and ciliate protozoa; the first are the most significant.
fertilizer	Inorganic fertilizers are plant nutrients prepared synthetically or mined as minerals. Organic fertilizers are sources of the same nutrients of animal and vegetable origins bound in organic form.
fetlock	The horse's 'ankle' joint in hind- and forelimbs between the metacarpus or metatarsus (cannon bones) and the first phalanx (long pastern bone).
FFA	See free fatty acids .
fibre	See crude fibre .
fibrin	The insoluble protein formed from fibrinogen by the proteolytic action of thrombin during normal clotting of blood.
filled legs	Oedema or puffiness of the legs. Abnormally large amounts of fluid (exudate) in the intercellular tissue spaces beneath the skin. The most common cause in healthy horses is a period of inactivity in a box following a season of hard exercise, particularly where corn or other concentrated feeds are being given. However, it is not protein poisoning. Oedema can also arise from diseases of the heart, liver or kidneys, or from long-standing malnutrition involving diets impoverished in protein.
flexor tendons	Muscles are attached to the long bones of the legs by extensor tendons, which extend or straighten the leg at that point when the muscle contracts, and by flexor tendons, which flex joints by contraction of the appropriate muscles.
founder, laminitis	A painful disease of the feet in which there is apparently a transitory inflammation followed by congestion of the laminae of the hooves. It is most frequent in ponies but can be induced readily in both horses and ponies by a sudden increase in the starch or protein content of the diet. Sometimes all four feet are affected, sometimes only the forefeet, and occasionally only the hind feet, or a single foot. Affected feet feel hot and the body temperature may rise. The stance is unnatural, affected forefeet are thrust forwards and the horse is reluctant to move.
free fatty acids (FFAs)	During work storage fats are mobilized when lipase enzymes catalyse the production of fatty acids, splitting them from glycerol and leading to a rise in the blood plasma concentration of both components.
frog	Horny central part of the lower surface of the foot, subject to thrush, an infection by bacteria and fungi occurring in wet unhygienic stables when there is poor routine foot care.

fundus gland region of stomach	A large region of the stomach containing glands of two types of cells. The region lies distal to the oesophageal and cardiac regions and proximal to the pyloric gland region.
fungal units	Fungi contaminating feed produce a mycelial mat of fine threads and fruiting bodies from which spores are shed. When badly affected feed is disturbed, the mycelial threads may break up into small particles and new growth can be initiated by each of these particles or by germination of spores. The fungal unit is any particle from which new growth can be started.
furlong	Forty poles, one-eighth of a mile. Originally the length of the furrow in the common field. The side of a square of 10 statute acres. Equals the Roman stadium, one-eighth of a Roman mile.
β-galactosidase	Neutral or brush-border lactase. This enzyme is present in the intestinal juice of normal young horses. It is necessary for the cleavage of milk sugar (lactose) to glucose and galactose, which can then be absorbed into the blood.
gamma globulin	A protein fraction of the plasma globulin, which has a slow moving electrophoretic mobility. Most antibodies are gamma globulins.
gastrointestinal tract	Stomach and intestines. Sometimes used synonymously with alimentary tract, which is the entire tube extending from the lips to the anus.
<i>Gastrophilus</i> GGT	See bot fly . Gamma-glutamyltransferase (EC 2.3.2.2) is released into the blood following liver damage. The blood activity of this enzyme is elevated in liver cirrhosis, pancreatitis and renal disease. The normal range of plasma activity is 0–41 iu/l and seneciosis may cause a rise up to 80–280 iu/l.
glossitis	Inflammation of the tongue. Observed in folic-acid deficient horses that have been treated for protracted periods with sulphonamide drugs (see 'Folic acid', Chapter 4).
glucagon	Polypeptide hormone secreted by the alpha cells of the islets of Langerhans in response to hypoglycaemia. It stimulates glycogenolysis in the liver and opposes the action of insulin .
glucogenic	Giving rise to, or producing, glucose. According to a long-used classification, amino acids are ketogenic if (like leucine) they are converted to acetyl-CoA and, when fed to a starved animal, produce ketones in the blood. Glucogenic amino acids such as valine, when fed to a starved animal, promote the synthesis of glucose and glycogen.
gluconeogenesis	The formation of glucose from amino acids within the body by various routes that include pyruvic acid and lactic acid, but not via acetyl-CoA.

glutathione peroxidase (GSH-P_x)	The activity of this enzyme (EC 1.11.1.9) in horse erythrocytes is used as an indication of the horse's selenium status. The activity is determined as μmol NADPH oxidized in 1 min by 1 ml erythrocytes. In TBs the evidence (Blackmore <i>et al.</i> 1982) indicates that the activity should be approximately 25–35 units/ml red cells.
glycolysis	The major pathway whereby glucose is metabolized to give energy is a two-stage process; the first stage, called glycolysis, can occur anaerobically and yields pyruvate.
glycosuria	The presence of abnormal amounts of glucose in the urine. It arises from failure of renal tubular reabsorption or from an abnormality of hormone status, as in diabetes mellitus.
goitrogenic	A term applied to substances in certain feeds, derived, for example, from members of the plant genus <i>Brassica</i> (family Cruciferae) which, if consumed persistently in large quantities, cause goitre (an enlargement of the thyroid gland). A deficiency of iodine in the diet will cause a similar condition, especially in young horses.
gossypol	A toxin found with two other toxic pigments in the pigment glands of cotton seeds. It is a polyphenolic binaphthalene derivative. It is inactivated by heating, but in this procedure it combines with lysine, reducing the protein value. Selective breeding has produced glandless seed, not widely available.
GOT	See AST .
GPT	See ALT .
grass sickness	A disease of horses, ponies and donkeys that seems not to be contagious. It occurs mainly in the summer months among grazing animals on certain pastures in Europe in a peracute form in which death occurs within 8–16 hours of initiation after some periods of great violence. In a subacute form the horse is dull, listless and salivates. The bowel becomes impacted and distended. Food material may appear in the nostrils. There seems to be some loss of motor control of the GI tract. Muscles of the back are hard, the horse is 'tucked-up' and twitching over the shoulders occurs. Recovery is uncommon (see Chapter 10).
growth plate haemagglutinins	See metaphyseal plate . Now called lectins, contained in <i>Phaseolus</i> spp., including <i>P. vulgaris</i> . Severe GI disorders can be caused by these substances when given in high dietary concentrations. They are proteins of which concanavalin A from jack beans is used medically as a mitogenic agent and preferentially to agglutinate cancer cells.
haematocrit	Packed cell volume (PCV). The proportion of blood by volume made up of cells, especially red cells, and expressed

haematopoiesis
haemoglobin

as a percentage. It is determined by centrifugation of blood samples containing an anticoagulant.

The formation and development of blood cells.

The oxygen-carrying red pigment of the red cells (erythrocytes) of blood. A conjugated protein consisting of the protein globin combined with an iron-containing prosthetic group (heme).

haemolysis
haemolytic icterus
of foals

The rupture of red blood cells.

Caused by the absorption of colostral antibodies that destroy the foal's red blood cells (see Chapter 7).

haustral

Referring to the haustra, or sacculations, of the colon.

haylage

Originally registered trade name for a silage containing a high proportion (35–50%) of dry matter made from wilted forage, precision-chopped to approximately 12–25 mm nominal length and ensiled in a Harvestore tower silo. However, the name has acquired a more liberal definition to include unchopped vacuum-packed material.

heating feed

A concentrated feed, which is readily digestible and fermentable and which leads to a rapid rise in blood metabolites, waste heat production and probably to some increase in metabolic rate.

heaves

See **broken wind**.

hepatic
encephalopathy

A degenerative disease of the brain resulting secondarily from advanced disease of the liver, or where a portocaval shunt occurs.

hepatitis

Inflammation of the liver.

hind-gut

The large intestine, consisting of the caecum and colon.

hives

Large numbers of small bumps, or raised areas, about 0.5 cm diameter under the skin, which eventually form scabs. They appear suddenly and are caused by an allergic response to specific components of feed, to drugs or to insect bites. The cause (**antigen**) can usually be determined by allergy tests, and recovery follows removal of the source from the diet or general environment. The condition sometimes arises when 'rich' feeds are suddenly introduced to the diet. The tarsal joint between the **tibia** and **metatarsus** (cannon bone) of the hindlimb.

hock**homeostasis**

Stability of the normal body states. Refers frequently to the constancy of pH and chemical composition of extracellular fluids.

hormone

A discrete chemical substance secreted into the body fluids by an endocrine gland and which influences the action of a tissue, or organ, other than that which produced it.

hotblooded horse

Hot- and warmblooded horses (cf. **coldblooded** horses) are

those derived to a significant extent from breeds originating in Mediterranean countries, which came to be called Arabian, Barb and Turk. Modern breeds of this type include TB, Arabian, Standardbred, American Saddle Horse, Morgan, Quarter Horse and Tennessee Walker. The principal North European warmblooded breeds are the Hanovarian, Trakehner, German Holstein, Dutch Warmblooded and Oldenburg breeds.

hyper-, hypo-	Prefixes: hyper signifying above normal or excessive and hypo meaning below.
hyperaemia	Engorgement with blood.
hypercalciuria	Abnormally large amounts of Ca in the urine; hypocalciuria is abnormally small quantities of Ca.
hypercapnia	Excess carbon dioxide in the body fluids.
hyperglycaemia	An elevated blood glucose concentration.
hyper- and hypokalaemia	Abnormal level of plasma potassium. Hypokalaemia may be caused by a combination of inadequate dietary K with excessive losses from the body. The causes of both hypo- and hyperkalaemia can be metabolic derangements, when there are abnormal shifts of K^+ between intracellular and extracellular space, as in acidosis.
hyperlipidaemia	A general term for elevation of any, or all, of the lipids in the plasma, including hyperlipoproteinaemia and hypercholesterolaemia, whereas hyperlipaemia refers specifically to an elevation of the triacylglycerols, previously known as triglycerides.
hyperparathyroidism	Abnormally increased activity of the parathyroid glands, causing loss of Ca from the bones.
hyperplasia	The abnormal multiplication, or increase, in the number of normal cells in normal arrangement in a tissue.
hyperplasmia	An excess in the proportion of plasma to cells in the blood.
hyperpnoea	Abnormal increase in the depth and rate of the respiratory movements.
hypersensitivity	An exaggerated reaction to a foreign agent. These immune responses are classified as immediate or delayed, or as Types I–IV.
hypertension	Usually refers to high arterial blood pressure, which may be confined to a specific circulation such as pulmonary or renal.
hyperthermia	Abnormally elevated body temperature.
hypertonic	A body fluid with a concentration, or osmotic pressure, above normal (more than isotonic).
hypertrophy	The enlargement or overgrowth of an organ or tissue by the increase in size of its individual cells.

hypervolaemia	In this text the term refers to red cell hypervolaemia. Hypervolaemia is an abnormal increase in the volume of circulating blood, which may be of plasma and/or of red-cell mass. In the studies referred to in Standardbred trotters, the plasma volume was within normal limits. The elevated volume of red cells should not be confused with an elevated mean cell volume (MCV), which is an abnormal mean increase in the volume of individual red cells, typically observed in alcoholism.
hypocalcaemia	A reduction of blood Ca below the normal range of concentrations.
hypocupraemia	A subnormal concentration of blood Cu.
hypoglycaemia	Concentration of blood glucose below the normal limit for the breed.
hypoglycaemic shock, insulin shock	Occurs when blood glucose falls below the normal range, causing nervousness, trembling and sweating.
hypomagnesaemia	A reduction of blood Mg below the normal range of concentrations.
hyponatraemia	Abnormally low plasma concentration of Na.
hypotension	Abnormally low blood pressure.
hypovolaemia	Abnormally decreased volume of circulating blood plasma.
hypoxaemia	Deficient oxygenation of the blood.
hypoxia	A reduction below physiological limits of the oxygen supply to tissues despite adequate perfusion by blood.
icterus	Jaundice. A yellowish discoloration of the visible mucous membranes – eyes, mouth, nostrils and genital organs. It can also be detected in blood plasma of stabled animals receiving a diet low in pigments and can be caused by certain infections resulting in destruction of the red blood cells and release of the haem pigment (haemolytic jaundice) or by liver damage.
IGF-I	See insulin-like growth factor-I .
ileum	The distal portion of the small intestine extending from the jejunum to the caecum.
immunity	Resistance to infection by an organism, or to the action of certain poisons. Immunity can be inherited, acquired naturally or acquired artificially.
immunoglobulin	Specific proteins, found in blood, colostrum and in most secretions, produced by plasma cells in response to stimulation by specific antigens, which in turn are inactivated. The antigens may be carried by, or released from, bacteria, viruses or even certain parasites.
imprinting	An inborn tendency of a neonatal foal to attach itself to a set group of objects, or a single object, such as its mother.

indispensable (essential) amino acids	Those amino acids that are not synthesized in the tissues of the horse, or otherwise made available from, for example, synthesis by gut microorganisms, in amounts sufficient to meet the requirements of tissues and which must therefore be present in the feed.
infarct	An area of tissue necrosis due to local anaemia resulting from obstruction of the blood circulation. In the text this refers to the effects of migrating strongyle larvae on the mesenteric blood vessels, which causes death of a segment of the intestines.
inorganic	Not a precise term but can be taken to refer to the ash content of the body remaining after it has been incinerated, which removes H, C and N as oxides. The minerals , in an oxidized form, remain, together with a very small proportion of carbonates.
insulin	A protein hormone synthesized by the islet cells of Langerhans in the pancreas and secreted into the blood where it regulates glucose metabolism. It is deficient in diabetes mellitus.
insulin-like growth factor-I (IGF-I) (somatomedin)	A protein with a topology resembling insulin and associated with its function, but produced by the liver and other tissues. Plasma concentration of IGF-I in man appears to depend on growth hormone (GH) secretion by pituitary tissue and on nutritional status. It stimulates protein synthesis and functions in cell differentiation and growth. IGF-I acts as an endocrine hormone via the blood and locally as a paracrine/autocrine factor, suppressing GH secretion and sensitizing insulin action.
international unit (iu)	As applied to vitamins, is the internationally agreed unit of potency for a particular vitamin that may have several molecular forms. The unit is now being displaced in favour of gravimetric measurements of each molecular form or vitamer.
intracellular space	The fluid volume within the cells of the body as distinct from the extracellular fluid space. Movement of ions, water, glucose, etc. from one to the other is under metabolic control.
intussusception	Prolapse of one part of the intestine into the lumen of an immediately adjoining part.
ischaemia	A deficiency in the blood supply to a tissue, organ or part of the body.
isoantibody	An antibody generated in the body in reaction to an isoantigen . An example is one found in the dam's blood and colostrum in response to a foetal protein that has entered her bloodstream.

isoantigen	An antigen in the body that will induce the production of an antibody against itself.
isoenzymes	Isoenzymes, or isozymes, are physically distinct, e.g. immunologically so, but they all catalyse the same reaction.
isotonic solutions	Those solutions that have the same concentration or, more specifically, the same osmotic pressure.
iu	See international unit .
jaundice	See icterus .
joint-ill	A disease in which organisms enter the body by way of the unclosed navel causing abscesses to form at the umbilicus and in some of the joints.
joule	SI (Système Internationale d'Unités) unit of energy: 4.184 J = 1 calorie. The joule is defined as the energy expended when 1 kg is moved 1 m by a force of 1 newton (1 Nm), $1 \text{ J} = 1 \text{ kg m}^2 \text{ s}^{-2}$, or 1 watt second (1 Ws) ($1 \text{ N} = 1 \text{ kg ms}^{-2}$). The kilojoule (kJ) = 10^3 joules and the megajoule (MJ) = 10^6 joules.
keratinize	To become horny. Keratin, the principal protein of epidermis, hair and the hoof, is very insoluble and contains a relatively large amount of sulphur. Increased keratinization of epithelia can occur under physiological conditions and under pathological conditions, e.g. vitamin A deficiency.
knuckling over	Usually refers to flexing of the fetlock joint owing to contraction of muscles and tendons or ligaments behind the cannon.
Kupffer's cells	Phagocytic cells lining the walls of the sinusoids of the liver and which form a part of the reticuloendothelial system.
labile	Chemically unstable.
lactase	An enzyme that hydrolyses the milk sugar lactose to form glucose and galactose. The enzyme accomplishing this in the intestine of the horse is β -galactosidase.
laminitis	See founder .
latent period	The period or state of seeming inactivity between the time of stimulation and the start of the response, e.g. the interval between the injection or absorption of antigen and the first appearance of antibody.
lathyrism	A condition characterized by sudden and transient paralysis of the larynx, with near suffocation of the horse, caused by β -aminopropionitrile in <i>Lathyrus sativus</i> (Indian pea) and other <i>Lathyrus</i> spp., including <i>L. odoratus</i> (sweet pea).
LDH	Lactic dehydrogenase (EC 1.1.1.27). A tissue enzyme that has several different forms or isoenzymes. It catalyses the transfer of H^+ with the formation of pyruvic acid and the reduction of pyruvate in the presence of NADH. The

	activity of LDH in the blood is elevated during and following strenuous exercise and following tissue damage.
legumes	Plants of the family Leguminosae or Fabaceae, which include useful forage species (e.g. the clovers and lucerne) and seed species (e.g. soya beans, peas and field beans)
leptin	A protein hormone produced by (white) adipose tissue and the pituitary gland, which acts on hypothalamic receptors to control food intake, as a satiety factor, energy balance and adiposity. In the horse, plasma levels are higher in fed than in fasted individuals and in fed animals plasma concentrations are higher during 12–24 hours than from 0–12 hours daily (Buff <i>et al.</i> 2001). In the human, and in other domestic and laboratory animals that have been investigated, these receptors may be down-regulated by luxury early nutrition or up-regulated by a scarcity of food. Down-regulation apparently increases the circulating levels of leptin (i.e. reduced receptor sensitivity) and the propensity to later obesity. The circulating level also changes diurnally with feeding, and, assuming a standard sensitivity, a raised level decreases energy intake. Genetic changes in the leptin molecule affect hypothalamic receptor sensitivity to leptin. In the text, low plasma leptin concentrations are correlated with anoestrus in mares.
leukocytosis	A transient increase in the number of white cells in the blood.
level of feeding	Weight of complete dry diet eaten daily, not confined to energy. Strictly it should be given as a proportion of metabolic body size ($BW^{0.75}$), or, more crudely, per 100 kg body weight.
ley	Grass and clover seeds sown as part of a cropping rotation, and usually ploughed-up after one to six years. The distinction from permanent pasture is not absolute.
ligament	A tough fibrous band supporting viscera, or which binds bones together. In different situations, ligaments are cord-like or in flat bands, or, in forming the joint capsule, they are in sheets, preventing dislocation.
light horse	A loose term for small and large lady's hacks, usually of mixed breeding, which may include TB and English Arabian. An alternative definition includes all riding horses of the present day in this classification, which excludes heavy draught horses and ponies.
lipases	A class of enzymes, members of which are found in digestive secretions and in body tissues. Individual lipases have as major functions the hydrolysis of fats to yield fatty acids,

	monoglycerides, glycerol and cholesterol, and the hydrolysis of phospholipids.
lipids	A group of substances found in plant and animal tissues, insoluble in water but soluble in common organic solvents, including petroleum, benzene, ether and chloroform. The crude fat of feed is the material extracted using light petroleum.
lipolysis	The decomposition of fat into glycerol and fatty acids and, in the case of, for example, the phosphoglyceride lecithin, into phosphoric acid and choline as well.
lucerne (alfalfa)	A legume, <i>Medicago sativa</i> . A perennial forage crop with a strong tap root, which grows well on light alkaline soils in warm climates. Weeds must be kept at bay during its establishment.
lymphatics	A system of vessels that drains lymph from various body tissues and conveys it to the bloodstream and that conveys neutral fats from the small intestine after they have been absorbed.
lysine	An indispensable amino acid , the concentration, or frequency, of which in most vegetable proteins limits their biological value. L-Lysine hydrochloride is sometimes used as a feed additive to make good any deficit in the dietary protein.
MAD fibre	Modified acid detergent fibre. A fibre fraction of feed determined by the MAD fibre procedure isolates principally the lignocellulose complex. This complex is the fraction of plant material that has most influence on energy digestibility of feed. In comparison, during the chemical procedure for crude fibre determination, a considerable amount of the lignin may become soluble and hence lost from the residue, so leading to an overestimation of the digestible fraction of feed.
maintenance	At the maintenance level of feeding, the requirements of the horse for nutrients for the continuity of vital processes within the body, including the replacement of obligatory losses in faeces and urine and from the skin, are just met so that there is no net gain or loss of nutrients and other tissue substances by the animal.
maize (corn)	<i>Zea mays</i> . A member of the grass family, the Gramineae, the seeds, both cooked and uncooked, of which constitute an excellent high-energy cereal grain for horses. The above-ground vegetative parts, at the milky-grain stage, can be made into a good silage for horses.
mandible	Lower jawbone. In the adult horse this bone has sockets for

	three incisors, one canine, three premolars and three molars on each side in the male (in the female the canines are usually absent, or rudimentary). Its grinding movements are controlled by powerful muscles.
ME	Metabolizable energy. The digestible energy (DE) of a unit weight of feed less the heats of combustion of the corresponding urine and gaseous products of digestion.
meconium	A dark-brown, viscid, semi-fluid or hard material which accumulates in the intestines of the foal prior to birth. It should be discharged soon after birth. The colostrum has a natural purgative action on it.
mesentery	Membranous peritoneal fold attaching the intestines to the dorsal wall of the abdomen.
metabolism	A term embracing the chemical processes of anabolism and catabolism in the body.
metacarpus	The part of the forelimb lying between the carpus (wrist) and the digit (<i>cf.</i> metatarsus). There are three metacarpal bones: the central, or third, metacarpal (cannon) is the largest; the other two are rudimentary metacarpals (splint bones).
metalloproteinase	Enzyme proteins binding a metal, including caeruloplasmin, cytochrome oxidase, lysyl oxidase and the superoxide dismutases.
metaphyseal plate	The region of linear growth of the long bones of growing horses lying between the epiphysis, or head, and the diaphysis, or shank.
metaphysitis	Inflammation of the metaphysis, seen as swelling or bumps adjacent to the joints of leg bones.
metatarsus	The part of the hind limb lying between the tarsus, or hock, and the digit. It is similar in layout to the metacarpus , with a central large metatarsal bone (cannon) and rudimentary metatarsals (splint bones).
methaemoglobin	A modified form of haemoglobin in which the iron has been converted from the ferrous to the ferric state in which it can no longer combine with, and transport, oxygen. The lesions can occur following the administration of large doses of certain drugs or after the consumption of nitrites in feed or water.
micronization	The cooking of cereal and legume seeds under ceramic burners that emit infrared irradiation in the 2–6µm wave-band, resulting in a rapid internal heating of the seed and a rise in water-vapour pressure, during which the starch grains swell, fracture and gelatinize.
minerals	The essential elements in the diet other than carbon (C),

hydrogen (H) and nitrogen (N). They include the macrominerals calcium (Ca), phosphorus (P), magnesium (Mg), potassium (K), sodium (Na), chlorine (Cl) and sulphur (S), and the microminerals iron (Fe), zinc (Zn), manganese (Mn), copper (Cu), cobalt (Co), iodine (I), selenium (Se) and fluorine (F). The microminerals are also known as trace elements. Some other elements, such as chromium (Cr), nickel (Ni) and molybdenum (Mo), are required in very small amounts. The mineral elements are required for incorporation into compounds different in the main from those in which they may appear in the diet whereas C, H and N are required as constituents of preformed organic nutrients. A crude approximation to the mineral content of the diet is obtained from the measurement of the **ash** content of the feed.

mitochondria

Minute bodies occurring in the cytoplasm of cells (except bacteria and blue-green algae, or cyanobacteria). They exert important regulatory functions both on catabolic and biosynthetic sequences. They are the seat of the citric acid-cycle, the β -oxidation pathway and of oxidative phosphorylation.

MJ

1000 kJ or 10^6 joules.

mycotoxins

Substances produced under specific conditions by certain fungi or moulds. Their chemical form and their effects on animals are wide ranging. These include hormone-like effects, disruption of intestinal, renal and hepatic function, tumour induction and antibacterial effects. Most antibiotics are mycotoxins. The best-known mycotoxins include: aflatoxin, produced by *Aspergillus flavus*; T-2 toxin, produced by *Fusarium* and *Myrothecium* spp.; ergotoxin, produced by *Claviceps purpurea*; zearalenone (F-2), produced by *Fusarium* spp.; dicoumarin, produced on melilots or sweet clover by *Aspergillus* and *Penicillium* spp.; and vomitoxin produced by *Fusarium* spp. Several mycotoxins are harmful to the horse in normal husbandry conditions.

myoglobin

A small oxygen-carrying protein of muscle tissue containing the pigment haem with an atom of iron at its centre. Severe muscle damage leads to its appearance in the urine in **azoturia**. Being a much smaller molecule than haemoglobin, it passes the glomerular filter much more readily, causing a dark-brown stain to the urine. Precipitation of myoglobin in the renal tubules, as with haemoglobin, may contribute to terminal **uraemia**.

myopathy	Disease of a muscle.
N-balance (N-retention)	The net gain of nitrogen (N) by the animal. $N \text{ in feed} - (N \text{ in faeces} + N \text{ in urine}) - \text{loss of ammonia expired in air per unit time}$.
NDF (neutral detergent fibre)	NDF is a measure of the indigestible structural carbohydrates of plant cell walls defined according to the method of determination of Van Soest (1963) and Van Soest <i>et al.</i> (1991) and described in Point 3 of Annex 1 to EC Directive 73/46/EEC(b). The procedure is also used in methods described in the booklet <i>Prediction of Energy Values of Compound Feeding Stuffs for Farm Animals</i> published by the Ministry of Agriculture, Fisheries and Food Publications, London SE99 7JT.
NE	Net energy, the energy value of animal product formed, or of body substance saved at or below maintenance, per unit weight of feed consumed. $NE = \text{metabolizable energy} - \text{heat increment}$.
necrosis	Death of a cell, or of a group of cells, which is in contact with living tissue.
NEFAs	Non-esterified, or free, fatty acids that are produced from the hydrolysis of triacylglycerols, or triglycerides.
nematode	The Nematoda are a class of tapered cylindrical helminths, the roundworms, of the phylum Aschelminthes. Not all are parasitic.
nephritis	Inflammation of the kidney; a focal or diffuse proliferative, or destructive, process which may involve the glomerulus, tubule or interstitial renal tissue (cf. nephrosis).
nephrosis	Any disease of the kidney, especially one characterized by degeneration of the renal tubules (cf. nephritis).
nephrotoxic	Toxic or destructive to kidney cells.
neurotransmitter	A chemical, e.g. norepinephrine, acetylcholine, dopamine, etc., released from the axon terminal of a presynaptic neuron on excitation, that travels across the synaptic cleft to excite or inhibit the target cell.
NFE	Nitrogen-free extractives. Measured in grams per kilogram of feed, this is numerically evaluated as $1000 - (\text{moisture} + \text{ash} + \text{crude protein} + \text{ether extract} + \text{crude fibre})$. NFE includes some of the feed cellulose, hemicellulose, lignin, sugars, fructans, starch, pectins, organic acids, resins, tannins, pigments and water-soluble vitamins if each of these components was present in significant amounts in the original feed.
non-protein nitrogen	See NPN .

NPN (non-protein nitrogen)	The determination of the crude-protein content of a feed assumes that the protein content is generally 6.25 times the determined N content. However, there are many compounds in feed, including nucleic acids, creatine and others, that have no protein value, but which are included in the calculation. Also, young herbage is rich in amino acids and nitrates that are components of NPN, although amino acids have an equivalent protein value.
nuts, cubes	Compounded horse feed mixtures that have been compressed into solid cylinders 3–10mm in diameter and two to three times as long by forcing the mixture through the holes of a metal die. The mix may or may not have been previously steamed in a kettle.
obligatory loss	Usually refers to the minimal inevitable loss of a nutrient, by excretion from the body, at low dietary intakes of that nutrient.
oedema	The presence of abnormally large amounts of fluid in the intercellular tissue spaces of the body. Applied in the text especially to an accumulation in the subcutaneous tissue.
oesophagus	The gullet, a muscular membranous tube or canal extending from the pharynx to the stomach.
oestradiol	The most potent naturally occurring ovarian and placental oestrogen which prepares the uterus for the implantation of the fertilized ovum and which induces and maintains the female secondary sex characteristics.
oestrous cycle	A cycle in the mare typically lasts 21 days in the breeding season in which there is a pattern of physiological and behavioural events under hormonal control. The cycle, which forms the basis of sexual activity, has two components: oestrus (heat) in which the mare is receptive to the stallion and the egg is shed, and dioestrus, a period of sexual quiescence.
oncotic pressure	As used in the text this refers to oncotic pressure of plasma, which is the osmotic pressure owing to the colloids present, principally albumin, that counterbalance the capillary blood pressure.
open knees	A dished concave appearance to the front of the ‘knee’ or carpus joint caused by epiphysitis immediately above the knee.
orad	Toward the mouth.
oral	Of the mouth.
organic	Complex molecules containing at least C and H and synthesized by living tissue.
osmolality	A solution that has 1 osmole solute dissolved in 1 kg water

has an osmolality of 1 Osm/kg (1/1000 Osm dissolved/kg water has an osmolality of 1 mOsm/kg; cf. **osmolarity**, the more practical measure, which is the osmolar concentration expressed as osmoles per litre solution rather than per kilogram water. For dilute solutions, as in the body, the quantitative difference is less than 1%.

osmolarity

The total number of dissolved particles, or osmoles, in water solution. Fluids with an osmolarity greater than that of body fluids are hypertonic and those for which it is lower are hypotonic. Osmolarity depends on molar concentration and not upon equivalents per litre (mEq/l). For instance, if Mg^{2+} concentration is 30 mEq/l, this is 15 mmol/l, or 15 mOsm/l. 100 ml 0.6 M NaHCO_3 provides 60 mmol Na^+ and 60 mmol HCO_3^- , or 120 mOsm, on the assumption that the salt is completely dissociated. The osmolarity of body fluids is about 285 mOsm/l.

osmotic pressure

The pressure that can be exerted when water moves from one solution to another of higher concentration through a semipermeable membrane, such as that of red cells. Solutions that have the same osmotic pressure, i.e. are isotonic with the osmotic pressure of red cells, are used for injections; otherwise, with the use of water, the cell membrane would become distended and burst (haemolysis). The energy of high-energy compounds is consumed when any nutrient is required to move against an osmotic pressure gradient.

osteoarthritis

Chronic, multiple, degenerative joint disease characterized by degeneration of the articular cartilage, hypertrophy of the bone at the margins and changes in the synovial membrane.

osteoarthrosis

Chronic arthritis of a non-inflammatory character.

osteochondritis

Inflammation of both bone and cartilage.

osteoid

The organic matrix of young bone.

osteomalacia

Softening of bones in adults from a deficiency of vitamin D or minerals. There is an increased amount of uncalcified bony matrix (osteoid).

oxalate

An organic acid anion that combines with Ca and some other positively charged dietary minerals to form a very insoluble precipitate that inhibits digestion and absorption (see 'Tropical grassland', Chapter 10). Oxalates circulating in the blood also react with ionized blood Ca and form a crystalline deposition in the kidneys.

oxygen deficit

O_2 deficit is defined by the O_2 equivalent of the difference between ATP supplied oxidatively from pulmonary $\dot{\text{V}}\text{O}_2$

- and that ATP utilized in the exercising muscle during intense exercise. A major portion of O₂ deficit in the horse arises from anaerobic glycolysis. Thus, the deficit is the O₂ required to fully metabolize the products of anaerobic glycolysis, supplied when intense exercise stops.
- Oxyuris equi*** Pinworm. A nematode intestinal worm, which is not a serious hazard to horses. It causes intense irritation of the anal region and this encourages tail-rubbing and biting. Piperazine compounds are effective.
- pancreas** A gland in the abdominal cavity that secretes a juice containing digestive enzymes into the duodenum (see Chapter 1) and secretes the hormones **insulin** and **glucagon** into the bloodstream.
- Parascaris equorum*** A nematode intestinal worm that commonly infects foals, yearlings and horses under three years old. It causes intestinal problems, colic, coughing and nasal discharge (see Chapter 11).
- parathormone**
parathyroid gland Parathyroid hormone synthesized by the **parathyroid gland**. An endocrine gland located in the upper neck adjacent to the thyroid gland. When serum concentration of calcium ions falls, parathyroid hormone is secreted into the blood. It induces mobilization of bone Ca, increases Ca absorption in the intestine, reabsorption of Ca by the renal tubules and the urinary excretion of P. Its effects are counteracted by that of **calcitonin**. See also **secondary nutritional hyperparathyroidism**.
- paresis (general)** A condition short of complete paralysis in which certain muscles are relaxed and weak. If it is more generalized the animal cannot support itself or stumbles. Sometimes there is slight paralysis – that is, there has been some injury to or effect on certain motor nerves and an inability to make purposeful movements.
- paresis (parturient)** This clinical sign, associated with **hypocalcaemia**, is *not* characteristic of mares. Lactation **tetany** was commonly observed in draught horse mares. **Hypocalcaemia** is a consistent characteristic, although **hypomagnesaemia** has been associated with recent transport. Mares grazing lush pasture and with a heavy milk flow are particularly prone to tetany. For other causes see Chapters 3, 9, 10 and 11.
- parturition**
pastern The act, or process, of foaling.
The region of the leg between the fetlock and the hoof in both fore- and hind limbs, formed by the long and the short phalanges which create the pastern joint. The third phalanx is in the hoof.

pelage	The hairy coat of the horse and other mammals.
peristalsis	The rhythmic involuntary muscular contractions of the alimentary canal by which it mixes and propels ingesta towards the rectum.
petechial haemorrhage	Small, subcutaneous blood spot caused by the effusion of a blood from capillary. Frequently numerous spots are present in tissue reacting to toxins, such as endotoxin.
pH	The symbol used in expressing the H ion concentration of water solutions. It signifies the negative logarithm of the H ion concentration in gram molecules (moles) per litre (the logarithm of the reciprocal of the H ion concentration). pH 7 is neutral. Progressively above this value alkalinity increases and below it acidity increases.
phosphagens	A group of compounds, including phosphocreatine and ATP, that occur in tissues and which yield inorganic phosphate with the release of energy on cleavage. They are classified as high-energy phosphate compounds.
photosensitization	The development of abnormally heightened reactivity of the skin to sunlight. In the text, reference is made to a skin reaction of horses following the consumption of certain plants. Reports exist of domestic animals reacting to St John's worts (<i>Hypericum</i> spp.) or bog asphodel (<i>Narthecium ossifragum</i>). White, grey or piebald horses or those with liver damage are most susceptible.
phytase	Phytase (EC 3.1.3.8) is produced by <i>Aspergillus niger</i> (CBS 114.94) and is listed as a permitted enzyme that hydrolyses phytates. Phytase is produced by many microbial cells, but, as far as it is known, does not occur in any of the digestive secretions of the horse.
phytates	Much of the phosphorus present in cereal grains and other seeds occurs as phytates, which are Ca, Mg, and other salts of phytic acid, a phosphoric-acid derivative, composed of a six-carbon ring structure with a phosphate group bonded indigestibly to each carbon atom. The horse can utilize the phosphate phosphorus only to the extent of about 30%, partly through the activity of bacterial phytase present in the intestines, but probably also as a result of some phytase activity which has been recognized in the wall of the intestine of several species. Phytates reduce the availability of dietary zinc. Wheat bran contains 1 % of P present as phytin.
piloerection	Erection of the hair.
pinworm	See <i>Oxyuris equi</i> .
pituitary gland (hypophysis)	A gland lying at the base of the brain and connected with the hypothalamus by the pituitary stalk. Physiologically it

consists of two parts, the anterior and posterior pituitary. Six hormones are secreted by the anterior portion: growth hormone, adrenocorticotropin, thyroid-stimulating hormone, prolactin, follicle-stimulating hormone and luteinizing hormone. The posterior portion secretes antidiuretic hormone (vasopressin) and oxytocin.

pK The symbol used in expressing the dissociation constant of weak acids (or bases) in the form of a negative logarithm. The larger the value the weaker, or less dissociated, is the acid. When equal concentrations of the salt of an acid and the acid are mixed, the $pK = pH$ and the buffering capacity of the mixture is maximal. Thus, for the primary ionization of carbonic acid to bicarbonate, as in blood, the $pK = 6.36$, and at $pH\ 6.36$ half the molecules of carbonic acid are dissociated forming bicarbonate. At the normal pH of venous blood (7.4) the mixture is an even more effective buffer to acid produced during anaerobic muscular activity with the formation of undissociated carbonic acid.

placenta An organ that develops within the uterus in early pregnancy and that establishes communication between the dam and the developing foetus. It is composed of a maternal portion and a foetal portion attached to the foetus by the umbilical cord. Following **parturition**, it is passed as the afterbirth.

progesterone A hormone liberated by the corpus luteum, adrenal cortex and placenta. It prepares the uterus for the reception, development and maintenance of the fertilized ovum.

prognathia Abnormal protrusion of the upper or lower jaw bone, creating difficulties, for example, in grazing.

prolactin A hormone secreted by the anterior pituitary gland that stimulates and sustains lactation.

prostaglandins Grouped in six main series of cyclic compounds derived from unsaturated fatty acids such as arachidonic acid – itself a derivative of the dietary indispensable (essential) linoleic acid – and from fatty acids with one less and one more double bond. Prostaglandins were first recognized in seminal fluid and the prostate gland. They show a variety of biological actions that influence smooth muscle contraction (as in contraction of uterine muscle and in blood-pressure control) and they are mediators in the regulation of the dilatation and permeability of arterioles, capillaries and venules in the inflammatory response. They are involved in immune mechanisms and are used for oestrus synchronization and for abortions in cases of twin foetuses. Prostaglan-

	din $F_{2\alpha}$ ($PGF_{2\alpha}$) which terminates the life of the corpus luteum has been the one most commonly used.
protease	An enzyme that digests proteins by hydrolytically splitting off amino acids. Several kinds are secreted into the alimentary canal (see Chapter 1).
protein	True proteins are chains in which the links are amino acids. All amino acids possess at least one N-containing amino-group. The crude protein content of the diet is defined as the N content $\times 6.25$ as it is assumed that protein contains 16% N. However, this method includes dietary nucleic acids, nitrogenous glycosides, amines, nitrates, etc. and so overestimates the true protein content.
proteolysis	The enzymatic digestion of protein, which, if carried out by the horse's own secretions, yields proteoses, peptones and amino acids, but, if carried out by intestinal bacteria, includes deamination with a loss of protein value.
prothrombin time	The synthesis of prothrombin occurs in the liver and requires vitamin K. It is essential for the clotting of blood, and any defect in prothrombin formation, or in the activity of other substances involved in clotting, extends the interval between the initiation of the process and the formation of fibrin from fibrinogen. Fibrin spontaneously coagulates. Dicoumarol, which arises from the activity of a mould on coumarin in spoiled sweet clover or melilots, interferes with the metabolism of vitamin K causing an extension of prothrombin time and consequential extensive haemorrhaging (see Chapter 11).
proximal	Nearest to the centre or origin, and opposed to distal. The duodenum is proximal to the jejunum and the 'knee' is proximal to the fetlock.
purgative	Cathartic, a medicine that stimulates peristaltic action and evacuation of the intestines.
pylorus	The distal or duodenal aperture of the stomach. It is controlled by a sphincter muscle and through it stomach contents enter the small intestine.
pyrexia	Abnormal elevation of body temperature.
Quarter Horse (American Quarter Horse)	A breed devised mainly from dams of Spanish origin, long bred by American Indians, and from Galloway sires, introduced by early settlers of North America.
quidding	The expulsion of partially chewed feed from the mouth. The habit may arise from injuries to the tongue, or cheek, resulting from molar teeth which are too sharp, irregular in height or in alignment, or even from permanent teeth pushing the temporaries out from the gums. Causes also include

	infections of the mouth or teeth and paralysis of the throat and consequent inability to swallow.
radius	One of the two long bones of the 'forearm', between the point of the 'elbow' and the 'knee'. The other long bone is the ulna.
ration	The amount of daily feed rather than its composition. It should include all the constituents of the diet apart from water.
rectum	The distal portion of the large intestine extending from the small colon to the anus and holding the faeces.
red blood cells (erythrocytes)	The most numerous cells in the blood ($6.8\text{--}12.9 \times 10^{12}/\text{l}$), there being only $5.4\text{--}14.3 \times 10^9/\text{l}$ white blood cells (leucocytes). The cellular portion of blood makes up 32–53% of the total, the remainder being the plasma. About 35% of each red cell is the protein haemoglobin, which transports oxygen from the lungs to the various body tissues.
renal clearance	The ratio of the concentration of a substance in the urine to that in the blood times the rate of urine formation. As the latter is normally unknown, the creatinine clearance ratio is measured (see Chapter 12). To calculate the glomerular filtration rate, when the rate of urine formation is known, creatinine or insulin may be used; these substances are readily filtered by the glomerulus but not secreted or absorbed by the renal tubules.
renal tubules	These run from the glomerulus through the cortex of the kidney, as convoluted tubules, then through the medulla as collecting tubules, and they open into the pelvis of the kidney at the apices of the renal pyramids. Their main function is the reabsorption of water and various solutes – glucose, chloride, Ca, P, etc. – required by the animal.
renin	A proteolytic enzyme synthesized by the juxtaglomerular cells of the kidney that plays a role in blood-pressure control by catalysing the conversion of angiotensinogen to angiotensin when renal arterial pressure falls.
reproductive cycle	The time from the conception of one foal to the conception of the next.
requirement (nutrient)	The requirement for any given nutrient is the amount of that nutrient that must be supplied in the diet to meet the net requirement of a normal healthy animal given a completely adequate diet in an environment compatible with good health. The net requirement is the quantity of that nutrient that should be absorbed to meet the needs of maintenance , including the replacement of obligatory losses, and of any work, growth, production or reproduction taking place.

respiratory exchange ratio	The respiratory exchange ratio is the $\frac{\text{rate of CO}_2 \text{ output by lungs}}{\text{rate of O}_2 \text{ uptake by lungs}}$
rheology	In many texts defined as 'R'. The science of the deformation and flow of matter. In this text it relates to the flow of blood through the vascular system, especially as affected by an elevated PCV during intense exercise.
rhinopneumonitis (equine)	A mild viral disease of the upper respiratory tract of horses, which also commonly causes abortion.
rickets	Defective calcification of the epiphyseal cartilage of growing horses owing to inadequate dietary vitamin D, Ca and P or an incorrect proportion of Ca to P in the diet.
ringbones	Any bony exostosis affecting the interphalangeal joints of the horse's foot, or any bony enlargement in the same region.
Ringer's solution	An isotonic solution, devised by Sydney Ringer, containing sodium chloride, potassium chloride and calcium chloride. However, it is but little more physiological than physiological saline as its chloride concentration is even higher and therefore it can cause metabolic acidosis of the same magnitude (see Chapter 9).
roughage	There are several types of roughage, which fall broadly into the following categories: (1) long and dry, e.g. hay and straw; (2) ground and pelleted hay, straw and oatfeed; (3) ensiled long grass and comparable succulent forages; and (4) chopped succulent ensiled material. Within each category only feeds analysed to contain more than 20% crude fibre on an air-dry basis should be included. Roughages tend to reduce the intake of dry matter in horses fed <i>ad libitum</i> and they decrease net energy intake in these animals in comparison with those also receiving concentrates . The fibre is useful in maintaining the microbial populations of the large intestine in a steady state.
ructus	Eructation or belching of gas from the GI tract.
ruminant	Herbivorous species that possess an enlarged forestomach (rumen) and that chew the cud by regurgitation of ingesta from the forestomach to the mouth.
sclerosis	An induration or hardening, especially resulting from persistent inflammation.
scours	Diarrhoea.
secondary nutritional hyperparathyroidism	The increased secretion of parathyroid hormone as a compensatory mechanism directed against a disturbance in mineral homeostasis induced by nutritional imbalances. A loss of Ca from the bones is induced, resulting in a condition

	marked by pain, spontaneous fractures, muscular weakness and osteofibrosis (see Chapters 3 and 11).
septicaemia	A serious condition in which bacteria circulate in the bloodstream and become widely distributed throughout practically every organ. The horse becomes distressed in severe septicaemia, respiration and heart rates are accelerated and body temperature is elevated.
serum	The clear liquid that separates from the clot and the corpuscles in the clotting of blood.
set-fast	See tying-up .
shank (of long bone)	See diaphysis .
silage (ensilage)	Succulent feed preserved either by adding acid or allowing natural fermentation to occur under anaerobic conditions in compacted material. The pH achieved is approximately 4–4.2. Too low a pH limits intake and too high a pH provokes protein breakdown. Materials ensiled include fresh grass, forage crops, the above-ground growth of young cereal crops and a variety of by-products from beet pulp to fish waste. The dry matter content is 30–50%.
spavin	Bone spavin is a disease of the hock, or tarsus, in which changes occur in the small bones on the inner aspect of the joint resulting in the deposition of new bone. Bog spavin is a puffy swelling of the same joint.
spleen	A gland-like but ductless organ in the anterior part of the abdominal cavity on the left side. Its functions are at least threefold: first, it disintegrates red cells, setting free the haemoglobin, which the liver converts to bilirubin, conserving the iron; second, it acts as a storehouse of red cells, which it releases into the blood during times of higher oxygen demand; third, evidence (mainly from other species) indicates a role for the spleen in immunological responses. The spleen is an antibody-forming tissue and macrophages constitute a predominant cell form in it.
splints	Bony enlargements that occur on the cannon bones or in connection with the small metacarpals or metatarsals (splint bones) as the result of localized inflammation of the bone or periosteum (periostitis or osteitis).
stifle	The joint corresponding to the human knee at the top of the hind limb.
stocking up	Swelling, or oedema , of the legs, owing to the accumulation of fluid beneath the skin frequently caused by a period of inactivity on rich feed immediately after an extended period

	of activity. Exercise, purging and a reduction in energy intake normally bring rapid relief. The condition is also occasionally seen in horses with damaged livers, especially where the diet is of low quality and deficient in protein.
strangles	An acute contagious fever of horses, donkeys and mules caused by the bacterium <i>Streptococcus equi</i> , characterized by catarrhal inflammation of the mucous membranes of the nasal passages and pharynx, and frequently accompanied by abscess formation in the submaxillary or pharyngeal lymphatic glands noticeable under the jaw.
stridor	A harsh, high-pitched respiratory sound.
stroke volume	See cardiac output .
strongyles	A group of strongyloid nematodes or roundworms widely distributed in the intestinal contents of mammals. In horses, strongyles are commonly called redworm (see Chapter 11).
sucrase (invertase)	A digestive enzyme secreted by the small intestine, which hydrolyses sucrose (cane and beet sugar), forming glucose and fructose, both of which are readily absorbed.
sweetfeed	An American term for a concentrate mix containing molasses.
synchronous diaphragmatic flutter	Contraction of the diaphragm in synchrony with the heart beat. It is observed in fatigued horses following severe exercise in hot weather when excessive sweating may precipitate a large decrease in the plasma concentrations of ionized Ca, Cl and K.
tachycardia	Excessive rapidity of heart action and pulse.
tachypnoea	Excessively rapid and shallow breathing.
tapeworm	A parasitic intestinal cestode composed of numerous flattened segments and attached to the gut wall by a head. Not infrequently occurring in horses, but generally causes no major problem. <i>Anoplocephala perfoliata</i> , the only species to have been observed infecting horses in the UK, may occasionally block the ileocaecal sphincter and cause severe colic.
tarsus	Bones of the hock. There are usually six bones of the hock, which join the tibia above to the two metatarsal bones below in the hind leg.
tetany	A condition in which there are localized spasmodic contractions, or twitching, of muscles (see Chapter 11 for stress tetany).
Thoroughbred (TB)	A hotblooded breed of about 1.625m (16 hands), which originated in the UK and which has been used to improve many other breeds.
thoroughpin	Distension of the sheath of the deep flexor tendon where it

	passes over the arch of the tarsus (hock) resulting from a sprain. It is not usually serious.
thrombophlebitis	Inflammation of a vein associated with thrombus formation.
thrush	A degenerative condition of the horn in the central cleft of the frog of the horse's foot caused by a bacterial infection and often resulting from the horse standing in dirty wet boxes with insufficient clean dry bedding.
thumps	See synchronous diaphragmatic flutter .
thyroid gland	Situated in the neck in connection with the upper extremity of the trachea. The gland secretes two hormones: iodine-containing thyroxin and calcitonin (thyrocalcitonin) (see Chapter 3).
tibia	The major long bone between the stifle and the hock .
tidal volume	The volume of air inspired or expired with each normal breath.
tillering	The process of forming side shoots from the base of the stem of graminaceous plants (cereals and grasses). Grazing, or cutting, in the vegetative phase of growth encourages this process, so thickening the 'bottom' or base of young pasture swards and increasing their suitability for grazing and exercising horses.
α-tocopherol	The principal tocopherol with vitamin E potency (see Chapter 4).
torsion	As described in the text, the twisting, or rotation, of part of the intestine, causing an obstruction.
toxin	Any poisonous substance of microbial, vegetable or animal origin.
trace elements	See minerals .
tricarboxylic acid (TCA) cycle	The series of metabolic reactions by which acetylcoenzyme A (acetyl CoA) is oxidized to carbon dioxide and water. The energy released is stored as ATP and the process occurs only in the mitochondria of cells (see Chapter 9). Acetyl-CoA is generated by the catabolism of fatty acids, carbohydrates and amino acids.
trypsin	This enzyme acts on peptide linkages that involve the carboxyl groups of lysine and arginine and ruptures protein chains at these points. It is one of the proteolytic enzymes secreted by the pancreas, but in the inactive form of trypsinogen. This is activated by the enzyme enterokinase liberated from the duodenal mucosa.
turgor	The normal consistency of soft tissue, as opposed to the flaccid condition of dehydration.
tying-up (set-fast)	A condition in racehorses in which stiffness, blowing and sweating occur after a period of hard extended exercise, said

	to be caused by a depletion of muscle glycogen (see Chapters 9 and 11).
tympany	Filling of the stomach or intestines with gas, usually caused by rapid microbial fermentation of ingesta, leading to a drum-like distension of the abdomen and colic.
ulna	A bone behind the radius in the foreleg that forms the point of the elbow. The shaft of the ulna is vestigial and the tapered end is fused to the radius.
umbilical cord	The nourishment of the foetus mainly passes to it through the cord from the placenta. After birth, the cord should not be interfered with as early severance deprives the newborn foal of 1000–1500 ml placental foetal blood, whereas under 'normal' conditions the amount is under 200 ml.
uraemia	The presence of urinary constituents in the blood and the toxic condition produced thereby. Normal blood urea level is greatly exceeded, indicating a failure of normal renal function.
urea	The chief nitrogenous waste-product synthesized in the liver, discharged from the body in the urine and also secreted into the small intestine. It is highly soluble in water and the amount produced by the healthy horse receiving regular meals is proportional to the crude protein content of the diet.
urticaria	See hives .
uterus	A hollow muscular organ lying in the female abdominal cavity below the rectum. In the mare it has a large body and small horns. It carries the foetal foal and nourishes it during pregnancy through the placenta attached to its wall.
vagus (pneumogastric) nerve	A major parasympathetic nerve (tenth cranial) of the autonomic nervous system possessing both efferent and afferent fibres distributed to the larynx, lungs, heart, oesophagus, stomach, liver, intestines and, in fact, most of the abdominal viscera. It therefore plays a considerable part in digestion and physical exercise.
vein	Thinner-walled vessels than arteries , in which deoxygenated blood, under lower pressure, is carried back to the heart. Veins possess a system of valves that control the direction of blood flow. Muscular contraction and relaxation of the limbs provides the main force by which blood is lifted back up the legs against the force of gravity.
vertebrae	A chain of bones running from the base of the skull to the tip of the tail and which carries in the spinal canal the spinal cord – the posterior part of the central nervous system.
VFAs	See volatile fatty acids .

villus (pl. villi)	Small vascular processes covering the mucous epithelium of the small intestine. They greatly enlarge the surface area for the absorption of nutrients into branches of the portal blood system and into the lymphatic system.
viscera	In this text the term refers to the abdominal viscera – the organs of the abdominal cavity.
vitamer	Any of a number of compounds that possess a given vitamin activity.
vitamins	A group of unrelated organic substances which occur in many foods in small amounts and which are necessary for normal metabolism of the body. They have been arbitrarily divided into a group of four major fat-soluble vitamins and at least ten water-soluble ones (see Chapter 4).
volatile fatty acids	Short-chain steam-volatile acids, principally acetic, propionic, butyric and smaller quantities of higher acids, which are the microbial waste-products of fermentation of dietary polysaccharides and protein within the alimentary canal. They are absorbed into the bloodstream and constitute a major energy source to the horse.
volvulus	Intestinal obstruction due to a knotting and twisting of the intestines.
warmblooded	See hotblooded .
wasting disease	A state of chronic emaciation.
wind-sucking (crib-biting, aerophagia)	In the English literature these terms refer to the act of swallowing gulps of air into the stomach. They are habitual vices. The crib-biter grasps the edge of the manger, fence, etc. with the incisor teeth and by the coincidence of the raising of the floor of the mouth, opening the soft palate and a swallowing action, a gulp of air passes into the stomach (Plate 11.1, p. 454). The wind-sucker achieves the same end without a resting place for the teeth. Young idle animals are said to acquire the habit from individuals in their company with a confirmed habit, which can initiate repeated bouts of mild colic. Excessive wear of the incisor teeth may compromise the individual's grazing powers. Sometimes a cribbing strap is fitted snugly around the upper neck of persistent offenders. The American literature restricts the term 'wind-sucker' to mares that aspirate air and faecal material into the vagina. This is corrected by Caslick's operation. The term ' cribbing ' is then reserved for the aspiration of air into the stomach.
withers	The ridge on the back of the horse over the dorsal processes of the thoracic vertebrae and the shoulder blades and directly in front of the saddle.

wobbler

The name given to a horse showing a slight swaying action of the hindquarters, or stumbling, occurring mainly between one and three years of age. The signs can become progressively worse over six to nine months when the horse is unable to trot without rolling from side to side and falling. The condition apparently results from damage to the spinal cord in the neck through injury and/or nutritionally induced abnormalities of the vertebrae caused by imbalances or inadequacies of Ca and P.

wood chewing

A habit developed by many horses probably as a result of boredom. The horse normally does not swallow the wood and the habit is unlikely to have any dietary implications.

**zone of thermal
neutrality**

The range of environmental temperatures over which heat production by the animal is minimized. Below this range the animal must increase heat production by shivering and other means in order to maintain a normal body temperature. Above the range, normal cooling mechanisms prove inadequate, body temperature rises and with it metabolic rate.

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Conclusion

The principles and science of equine nutrition and feeding practice can still be accommodated within one text, yet the saga is incomplete. Completion is never likely, as our environment is continually evolving. This evolution will necessitate the unceasing adaptation of equine husbandry and the prescient anticipation of likely needs in the decades to come. For many, the unfortunate contraction of agricultural production in western Europe may allow pastures to be developed specifically for horses, although, for many stables, access to adequate pasture will continue to decline. Both these developments are attended by questions that need addressing and our research. There is now a wider use of haylage than there was in the 1990s and reference is made in this text to improved analytical procedures that more reliably describe forages. This facility for descriptions of greater relevance should allow a more economic use of land and should be an asset to better husbandry, leading indirectly to a reduction in the risk of metabolic diseases related to the GI tract. In contrast to the situation in the 1990s, there is presently much greater illumination of the relationship between diet and endocrine secretions. This may ultimately assist guidance towards better control of reproductive physiology and obesity. Yet in several areas of practical husbandry there is only an inchoate understanding of the physiological principles. Hence, skill and experience in feeding practice must reign in the management of individual horses; but I hope that the present text will provide a useful summary of recently published scientific evidence to be accommodated by readers with these skills.

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